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SOMATICOVISCERAL FUNCTIONAL DISORDERS FROM EYE-STRESS

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Previous to two decades before this writing, hundred-weights of literature had been published on the causation of the numerous organic diseases of the eye by diseases elsewhere in the body; and since the popularization of the doctrine of focal infection, this literature has been added to by tons.

But the other side of the picture, how much functional disturbance elsewhere in the body can originate from the eyes, has not been given the recognition that its importance deserves.

Some phases of this subject have been set forth in my previous papers.[1]

Aside from giving due recognition to the late Dr. George M. Gould for the great pioneer work done in this field, no effort will be made to give all references to authorities quoted, or to compile statistics, and figures and formulas will be cut down to the fewest possible.

As knowledge advances, the tendency to consider visible gross or microscopic pathology the result of physiology gone wrong increases, and much of recognized bacterial infection will not take place unless the threshold of physiologic resistance is lowered by something which is usually invisible.

Two basic systems of living structures are circulation and innervation of cells, and whatever disarranges either of these can disarrange the function of organs, and whatever disarranges the function of organs gives rise to symptoms.

Maitland Ramsay showed, in the *Glasgow Medical Journal*[2] and in the *Lancet*,[3] the similarity of the innervation of the ciliary muscle and iris, the heart and the intestinal tract; hence, the connections by which the former can influence the latter. It will probably be admitted as an axiom that an automatic vital mechanism will continue to work

1. Jones, E. L.: Disturbances of the Heart and Liver by Low Grades of Astigmatism, Tr. Am. Acad. Ophth., 1919, p. 209; Eye Stress as a Cause of Neurocirculatory Asthenia and Other Functional Heart Disorders, Am. J. Ophth. **6**:746, 1923; Disturbances of the Autonomic Nervous System by Eye-Stress, Internat. Clin. **1**:213 (March) 1928.

2. Ramsay, A. M.: Glasgow M. J. **106**:337, 1926.

3. Ramsay, A. M.: Lancet **2**:1063, 1931.

properly unless some interference takes place, until something essential wears out. In the human body, these interferences are so numerous that all of them could never be listed.

The autonomic nervous system and the ductless gland system occupy the highest stations so far attained in the effort to reach the mystery of life, and whether they are equal copartners or one dominates the other is not yet known. But it is known that whatever disturbs either system can have varied and far-flung manifestations.

It is the contention of this paper that eye-stress affects the autonomic nervous system in susceptible patients. Hence, when the autonomic nervous system is interfered with, the basic functions of circulation and cellular innervation are interfered with, and a regular Pandora's box of troubles may be opened.

There are many other things beside eye-stress that disturb this same autonomic nervous system and open up the same Pandora's box; so that most of the symptoms which will be ascribed to eye-stress also occur in the domain of practically all the subdivisions of medical practice.

The syndrome of headache, dizziness and pain in the neck and shoulder, often going to the finger tips, with numbness or tingling and paresthesia, I have recognized for at least a quarter of a century as a manifestation of eye-stress; but in that time, it has also been claimed by the sinuologists (Sluder's syndrome), by the urologists as pointing to kinks in the ureter, by internists as indicating focal infection, and probably by podologists as a sign of fallen arches. And at times all of them may be right.

The splanchnic viscera, when ill at ease, are apt to cause bad dreams or nightmares, or crying out in sleep, and no line is drawn between overloading the stomach with indigestible food before retiring and eye-stress as the cause.

Many years ago I was impressed with the number of persons who ceased to have bad dreams and nightmares to a marked degree after having eye-stress corrected.

One patient, a middle-aged man of noble and generous nature, when asked if he had bad dreams, laughed and said that he did nothing in his sleep except run from the devil. Some months after having the vision of his only seeing eye properly corrected, he was asked about his dreaming, and said, "The devil has entirely ceased chasing me."

Eye-stress stands in the front row as a thief of nervous energy, and every patient with nervous or physical exhaustion not clearly due to other causes should be given the benefit of a thorough examination, as should every person suffering from dizziness not due to evident causes. It is not usually the patient with large errors, and seldom the one with substandard vision, who suffers eye-stress, but one who has

normal or supernormal sight either without glasses or with the ones that he is wearing. Relief from stress is not attained by giving the best vision possible, but by exact correction of astigmatism.

The late Dr. Julian J. Chisolm of Baltimore, who was ahead of his generation in many ways, contributed an article to the Section of Ophthalmology of the American Medical Association in 1888, entitled, "The One-Quarter Dioptric Cylinder Lens, the Most Useful of the List of Cylinders," and his last listed publication, in 1894, was, "Degrees of Astigmatism, However Low, When They Annoy Should be Corrected."[4]

My motto is: "In the presence of symptoms, correct the smallest degrees of astigmatism, alone or in combination with spherical lenses, and correct the larger degrees with such accuracy as not to leave any residual astigmatism to cause stress." Many think that in the presence of strong spherical errors, a little astigmatism does not matter, but experience has shown that some of the most startling results can be obtained in these cases, in which the strong sphericals alone have fallen short of giving the benefits derived from the small added cylindric correction.

It is also absolutely necessary to wear the glasses all the time to get results. In young people it is a matter of days only, in middle-aged people a matter of weeks, and in old people usually a matter of months before nature can readjust the mechanisms that have been out of order; to leave the stress-removing glasses off at times prevents the readjustment from taking place. The rule does not apply to glasses that are only sight-giving; sight-giving is the limit of the layman's conception of the function of lenses.

The program of societies devoted to the eye, and of the ophthalmologic sections of general societies, usually consists of papers dealing with the surgical or medical sides of ophthalmology, with an occasional exception.

One of the most notable exceptions in years was the presidential address to the Ophthalmological Society of the United Kingdom a few years ago by Mr. Ernest Clarke, of London, who had been previously known by his textbook and other contributions as a thorough, painstaking refractionist.

He set forth with great clearness many things in the body that can come from so-called eye-strain, and emphasized the necessity of correction of small errors and exact correction of larger ones.

It is easy to get the best vision-giving formula for lenses from test cards containing letters from the largest to the smallest used for distance, or from the multiple light cabinets of various designs,

4. Chisolm, J. J.: Tr. Internat. Ophth. Cong., Edinburgh **8**:285, 1894.

but what I have found most useful for getting the stress-relieving formula is a small single light box the aperture of which shows only one line at a time and is worked by an overhead cord on pulleys; the final testing is done on the smallest single square letter that can be seen, and the cylinder axis is set midway between the points where first blurring shows when rotated in each direction from the supposed axis. With weak cylinders, this will fix an axis much more accurately than trying to tell where vision is best. And on this exactness of axis, as well as the dioptric strength of lenses, rests the difference between success and failure.

I am fully aware of the condemnation that has filled textbooks and current literature for several decades on the use of cycloplegics or mydriatics beyond certain ages, but I have also found out the impossibility of getting the stress-relieving formula, especially the exact cylinder axis, without their use; in the presence of serious symptoms of stress, I have used them at ages much beyond established standards, of course always taking the tension first, and avoiding them in cases that show any increase. Where age has almost nullified the spherical changing power of the ciliary muscle, the minimum of homatropine that will induce slight mydriasis is used when the pupils are not 4 mm. or so in diameter to prevent the stenopeic action on light rays, which can totally falsify the most careful tests; I then contract the pupils at once by means of physostigmine.

This paper could be prolonged *ad infinitum* by the narration of cases, but only such cases will be used as are necessary to illustrate all phases of the subject, with a little added emphasis on the subject of frequent or continued crying whether the patient is awake or asleep, which to my knowledge has not been described elsewhere.

While it is quite common to note more than one of the types described in this paper in a family, or in two generations of the same family, I wish to declare my belief that it is not the type of eye that is transmitted by inheritance, but the type of nervous system that will react to the deleterious effect of eye-stress. There are probably more persons with astigmatism of varying degree who never have any symptoms than there are of those who do, and some astigmatism exists in practically all eyes.

Some of these patients have been followed from their "teens" into the age of presbyopia, and the susceptibility remains, as evidenced by the recurrence of the same symptoms after from one to many years, and by relief when changes are corrected. Two things in this respect cause wonderment. The first is the small change in some people, often in one eye only, that will cause a return of symptoms, and the second is the great change in some persons, even in both eyes, that may be necessary before the symptoms recur.

The intent of this paper is to emphasize the relation of eye-stress to general bodily conditions, but in narrating cases, the matter of ocular discomfort or visual trouble must be mentioned, as that is what brings the patient to one for examination. He not only does not suspect that the general symptoms come from the eyes, but is skeptical and sometimes openly antagonistic when this is explained, and even after relief is obtained, has his own explanation.

REPORT OF CASES

CASE 1.—A woman of about 72 years was seen because of certain minor discomforts in the use of the eyes. She had strong spherical bifocal glasses, which gave full vision far and near.

On questioning her in the presence of her middle-aged daughter, I found that she had had gaseous eructations for over forty years for which she took soda or other medicine several times every day. A careful test showed a small amount of astigmatism in addition to a large degree of hyperopia, and the opinion was ventured that this might have something to do with the formation of gas in the stomach.

She was not seen again for three years; then the daughter brought her back, stating that her mother had said to her, "I must go to the doctor and get some medicine; my stomach is beginning to gas again." The daughter answered, "You took medicine for forty years and nothing did you any lasting good until you got your eyes fixed. We will go and find out if your eyes have changed; if they have not, we will then go to see the doctor." A retest showed that changes had taken place in the patient's astigmatism.

Some of these patients who feel so miserable, weak and helpless look the picture of health, and must rest under the imputation of feigning illness to shirk their duties and obligations; while others, feeling no more miserable, weak and helpless, look so frail, thin and colorless as almost to make their friends weep in sympathy.

An illustration of the latter type of case follows:

CASE 2.—Mrs. E., aged 42, the wife of a minister in a rural region, consulted me on Aug. 8, 1919, because of commonplace ocular symptoms of the kind mentioned. She was a pale, thin, spiritless woman, a "bundle of nerves," depressed and too weak to work; for many years she had suffered from gaseous eructations and dizziness, which kept her in bed as long as six weeks at a time; while of average height, she weighed only 85 pounds (38.5 Kg.).

She was wearing weak spherical lenses, obtained in a state where she formerly lived. As is often the case in patients whose reserve nerve force is exhausted, retraction was extremely difficult, as vision could not maintain itself for any length of time, and as the patient's answers varied greatly or were contradictory. Much time and patience had to be expended before a formula could be established. There was an amblyopia of 20/50 in one eye in which the error was quite a large degree of astigmatism; in the other there was astigmatism of 0.62, with a weak spherical lens.

Two years later the patient's husband reported that she was much better in every way, and was gaining in health, weight and happiness.

On Sept. 9, 1924, she returned because of presbyopia, and was not recognizable as the same person. She had increased from 85 to 125 pounds (56.7 Kg.) in weight, had good color and spirits, and looked many years younger.

The husband was subsequently transferred to a remote state. On June 8, 1929, the patient brought a 20 year old daughter to have her eyes tested. She had continued to hold her improved health, and looked many years younger than she did ten years before, and might easily have been taken for a sister of her daughter.

All tests are first made without cycloplegia, and in a minority of instances give the correct formula, but in a large majority there would be sufficient difference to lower the result very much from the standpoint of stress-relieving—not from the standpoint of sight-giving.

That age is no barrier against these malevolent influences is borne out by the reports of several patients past 70 years of age, especially in the matter of enfeebled heart action, dizziness and gas-formation in the stomach. Some of these patients report that they seem to be "going stronger" at 75 than at 65.

Illustrative of this is the following case.

Case 3.—Mrs. N., aged 69, a superadipose person, had been treated by her local physician for two years. Shortly before reporting to me for examination she had gone to a large city to undergo a complete study by physicians connected with a famous university. No definite findings were made, and she was told to enter the hospital for further investigation. As no bed was available without many days' waiting, she came home.

The fact that she had no visual comfort, in spite of having seven pairs of glasses for near use only, gave the opportunity to study her from the standpoint of eye-stress.

She was anemic, had suffered from headaches all her life, was extremely drowsy and indifferent to her surroundings and immensely depressed. She was easily tired and winded and had suffered from gaseous eructations for many years, with cardiac oppression and palpitation, severe insomnia, startled awakening and internal tremor. She was so dizzy that in crossing the street she reeled like a drunken man and caught with her hands any pillar or post available. For the past eleven years, she had fallen so frequently that her body was constantly covered with bruises.

Under minimal mydriasis, not cycloplegia, her astigmatism was fully corrected.

Four months later, she was met by chance on the street and reported herself much better.

Nearly two years after her first visit, she returned saying that owing to breakage she had been going without the glasses for far vision and her symptoms were returning. The glasses were replaced and she subsequently adopted bifocals.

Five years later, she reported that she was in better health than she had been for many years, had no dizziness and little trouble of any kind, had plenty of energy and interest in life, and but for the fact that her thigh bone had been broken in an accident, which crippled her, would be as active as in the days of her youth. Her anemia had disappeared.

I was astonished at how much younger and more vivacious she looked than when I first saw her.

Many people go through life trying all schools of healing which fail to give lasting relief from symptoms originating in eye-stress. A

perusal of newspaper testimonials as to the most advertised cure-all combination liver, stomach, bowel and nerve regulator gives a graphic description of them, but the relief does not last. By reason of the frequency of the pain or numbness in the neck, shoulders, arms and fingers, the osteopaths find this a happy hunting ground, as their pulling and pounding give temporary relief, but it does not last. Some patients say that taking so many of these treatments keeps them financially bankrupt, and they welcome any measure that will give lasting relief and liberate them from the thralldom of having to pay, pay, pay.

In the next case the patient came to see me about an ear that had been deafened when he fell while standing still and struck the back of his head on the concrete pavement; bleeding from the ear and nose had followed. As a basal fracture was inferred from the fact that the hearing was dull and the canal was filled with clotted blood, the patient was sent home to bed and magnesium sulphate was prescribed; he was instructed to send for his family doctor should other symptoms develop.

After his recovery, he returned on Jan. 30, 1925; his record says:

CASE 4.—Mr. W., aged 61, for the past eighteen years had had mild epilepsy, falling from two to six times a year. He had most of the commonplace local symptoms of eye-stress, including headaches from his earliest recollection; he was often sick; severe dizziness had occurred over twenty years; pain in the neck and shoulders extended to the finger tips, with numbness, and sometimes to half of the body; aphasia occurred often; he had cold, damp hands and feet, and easily became tired and winded; gas formation in the stomach had been noted for twenty years; he complained of insomnia, bad dreams and startled awakening.

Under the minimal mydriasis, his error was found to be O. D. $+0.75 \supset$ cyl.$+$ 0.50 ax. 105; O. S. $+0.75 \supset$cyl. $+$ 0.50 ax. 15; this correction was given in bifocals.

Before the end of a year, he had become practically rid of all symptoms, had gained 18 pounds (8.1 Kg.), and had improved so much in general appearance that it was a matter of pride with him to hear his friends tell him how much younger he was looking.

During his unconsciousness from the falls that he had suffered for eighteen years, he frothed at the mouth, bit his tongue and had convulsions.

The last note on him is that of Feb. 17, 1928, when he came in because of broken glasses. There had been only one falling spell since he had his eyes corrected; this occurred at the end of the eleventh month.

This patient was presented before the Maryland, Virginia, West Virginia and District of Columbia subsection of the American College of Surgeons when it met in Cumberland, Md., and the title of the report was, "Disturbances of the Autonomic Nervous System by Eye-Stress."

The proffered explanation of many of these symptoms lies in the vasomotor control of the arterial system supplying the brain centers, and varying according to whether the vessels are too much relaxed and flooded, or by spastic contraction made anemic.

Several dozens of patients who had undergone removal of the appendix or operations on the gallbladder, which relieved them of most of their symptoms, were left with the hangover of gaseous eructations, often worse than before the operation; this was entirely cured by proper stress-relieving corrections, even though many of the patients already had glasses giving 100 per cent vision, or had 100 per cent vision without glasses.

In one or two cases, in which the appendix had been removed solely to stop the formation of gas, with no benefit, the patient found relief in ocular correction. Of course, the reverse of this also occurs, when the oculist hopes for relief not obtained until the surgeon removes the appendix or the gallbladder.

In 1929, Lebensohn [5] described in the *American Journal of Ophthalmology*, under the title of "Oculovisceral Reflexes," experiments conducted on himself in the physiologic laboratories of the University of Chicago, in which he swallowed a small rubber bag attached to a tube projecting from the mouth.

After a proper degree of inflation to adapt the bag to the stomach cavity, tracings of the gastric musculature were made on a revolving drum by a needle connected with the tube. He then made his eyes astigmatic by means of lenses, and continued reading; the tracings showed quick disorder in the stomach muscle, thus objectively demonstrating what had long been observed in a clinical way.

According to my understanding of the term in general use by the public, the condition called "nervous indigestion" has almost invariably been wholly or largely relieved by exact correction of any astigmatism present and constant wearing of glasses. Yet in a recent work on nervous indigestion by an author high in medical circles, only nine lines in two hundred and eighty-four pages are devoted to the eyes as a causative factor.

Anemia is not rarely one of the results of the disturbance from eye-stress, as illustrated by the following case:

CASE 5.—Sadie M., aged 20, was brought from a mining town on Feb. 17, 1930. She was pale and anemic; in addition to local ocular symptoms, she suffered from dizziness, pain in the neck and shoulders, low spirits, extreme nervousness and depression and gaseous eructations; she easily became tired and winded, and had experienced pain in the side from running since early life; she awoke, startled, with cardiac oppression and rapid breathing, and had clammy, cold feet; for several months she had continued spells of causeless and unexplainable crying.

The refractive errors were corrected by glasses for constant wear, and so quick was her improvement that in less than three weeks the local newspaper correspondent reported her as rapidly recovering her health.

5. Lebensohn: Am. J. Ophth. **12**:562 (July) 1929.

She came in with another patient on Aug. 16, 1930, and was a picture of health and happiness, with splendid color, physique and mental alertness which continue up to the time of writing.

Timidity, lack of self-confidence and senseless crying in children and some women with morbid fears as of sudden death are often due to the depressant effects of eye-stress and are relieved by proper cycloplegic correction, which may change the patients' whole course of life to a higher plane. In some men the crying takes place during sleep only.

In these cases "crying" means the shedding of tears; crying out in sleep means only loud use of the voice and is found from early youth to late old age. In one instance, a widow in her forties slept in the same bed with her mother in her seventies, and they took turns in sleeping because the ranting one always kept the other one awake. Proper corrections in each case almost entirely stopped this habit and improved the general health in both, as reported four years later.

In my belief, such cases explain many of those which psychiatrists classify as "anxiety neuroses," manifested by psychic conflicts, touchiness, irritability, sensitiveness, fear of death, etc.

The sweat glands are known to be under autonomic nerve control, hence the frequency of sweating hands and feet when the autonomic nervous system is thrown out of adjustment.

CONCLUSION

It is the contention of this communication that by absolutely exact correction of astigmatism, the glasses being worn constantly, one can get results impossible of attainment by corrections that may give the best vision but do not relieve the stress.

CHEMISTRY OF THE LENS

I. COMPOSITION OF ALBUMINOID AND ALPHA CRYSTALLIN

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In this article the chemical analyses of two of the proteins of the bovine lens are described. The analyses of these proteins will probably be similar to those of the proteins of the human lens. Until more chemical information of the normal lens protein is available, it will be difficult to interpret the chemistry of the cataractous lens. The problems of the formation of the cataractous lens are many, and the amount of material is small.

The investigation of the lens protein was begun in 1830 by Berzelius,[1] who called the protein of the lens, crystallin or globulin. Later, Mulder,[2] Rüling,[3] Lieberkühn,[4] Vintschgau,[5] Béchamp,[6] Laptschinsky[7] and Cahn[8] studied the lens protein without adding any information that may be used at the present time. Mörner[9] was the first to analyze the proteins systematically. Jess[10] followed Mörner with more detailed investigation of these proteins. The articles of Mörner and Jess will be discussed later in this communication.

From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

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9. Mörner: C. T.: Untersuchung der Proteïnsubstanzen in den lichtbrechenden Medien des Auges, Ztschr. f. physiol. Chem. **18**:60, 1894.

10. (*a*) Jess, A.: Die moderne Eiweisschemie im Dienste der Starforschung: I, Arch. f. Ophth. **105**:428, 1921; (*b*) Die Monoaminosauren der Linsenproteine, Ztschr. f. physiol. Chem. **110**:266, 1920.

PREPARATION OF THE PROTEINS

Material.—Bovine eyes were obtained immediately after slaughter and were used at once. The lenses were removed through a corneal incision. The capsules were removed from the lenses, and the lens substance was macerated mechanically in distilled water, 5 cc. of water being used for each lens, until no fibers were found microscopically. The solution of lens protein was centrifugated to remove the insoluble albuminoid. The albuminoid was washed with distilled water until the wash water gave no test for protein. It was washed with alcohol, then ether, and dried at 105 C. Extractions of albuminoid with solutions of sodium chloride in various low concentrations gave no soluble protein.

The supernatant liquid containing the other proteins was slowly passed through a Sharples supercentrifuge and then through a sterile fine Berkefeld filter. The clarified solution amounted to about 10 cc. for each lens used. Ten per cent acetic acid was added until the maximum precipitation occurred. The amount of acetic acid that was added was determined by trial in test tubes. The p_H of the liquid, determined colorimetrically, was 5.2. The precipitate was alpha crystallin. It was centrifugated off and suspended in water. A two normal solution of acetic acid was added to the mixture until the alpha crystallin just passed into solution. The p_H of the solution was 4.6. The protein was reprecipitated at p_H 5.2 by the addition of one-fifth-normal solution of sodium carbonate. The acid was used to dissolve the protein in order to avoid any chemical change caused by alkalis. The coagulation point of 1 per cent solution of alpha crystallin in 5 per cent solution of sodium chloride at a p_H of 6.8 was 72 C. If coagulation of other proteins occurred in the test, the protein was redissolved and reprecipitated until only alpha crystallin was present. After washing with 95 per cent alcohol and then ether, the protein was dried at 105 C. From a 0.37 per cent freshly prepared solution of alpha crystallin at 25 C. and p_H 6.8, the precipitation of this protein began at 15.2 per cent and was complete at 24.6 per cent saturation with ammonium sulphate.

The solution remaining after the first precipitation of the alpha crystallin was neutralized with a fifth-normal solution of sodium carbonate to p_H 7.2, and then dialyzed in small collodion sacs with distilled water at 0 C. for four days. A large part of the beta crystallin was thus precipitated. The protein was centrifugated off, redissolved and reprecipitated in the same manner. The coagulation point of 1 per cent solution of protein in 5 per cent solution of sodium chloride at a p_H 6.8 was 61 C. No other protein was found present by coagulation. The protein, after being washed with 95 per cent alcohol and then ether, was dried at 105 C. Beta crystallin in 0.5 per cent solution was found to precipitate in from 19.76 to 33.44 per cent saturated solution of ammonium sulphate at p_H 6.8 and at 25 C.

The supernatant liquid freed from the beta crystallin was saturated with sodium chloride. A small amount of protein was precipitated, which gave coagula at 72, 62 and 52 C., indicating that it consisted of alpha crystallin, beta crystallin and albumin. After this protein was removed and discarded, the solution was then made acid with acetic acid until a maximum precipitation occurred. The precipitate that consisted of albumin was filtered off. The filtrate gave no biuret test for protein. The albumin was redissolved in a small amount of water and reprecipitated in the foregoing manner. It was washed with 95 per cent alcohol and then ether, and dried at 105 C. The coagulation point of about 1 per cent albumin in 5 per cent solution of sodium chloride was 53 C.

A second method for the isolation of beta crystallin and albumin was also used. After the first precipitation of alpha crystallin, the solution containing the other two proteins was neutralized with two normal sodium carbonate, and then half saturated with ammonium sulphate. The large precipitate that formed consisted of beta crystallin mixed with a small quantity of alpha crystallin. After the precipitate was redissolved in water and dialyzed for five days in distilled water, the beta crystallin was precipitated. It was centrifugated off and washed repeatedly with distilled water. The centrifugate of wash water was discarded. The supernatant solution, after the removal of beta crystallin, was then saturated with ammonium sulphate. The precipitate of albumin that formed was filtered off, redissolved, dialyzed and then coagulated by heat. After washing with 95 per cent alcohol and then ether, it was dried at 105 C.

ANALYSES OF THE PROTEINS BY THE VAN SLYKE METHOD[11]

Duplicate samples of about 2 Gm. of each of the proteins were hydrolyzed by boiling for about thirty hours with 150 cc. of 20 per cent hydrochloric acid. The phosphotungstates of the bases were decomposed by the amyl alcohol-ether method.[12] The distribution of nitrogen in albuminoid and alpha crystallin is shown in tables 2, 3 and 4. The amino-acids in the protein, as calculated from the Van Slyke and colorimetric analyses, are given in table 5.

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11. (*a*) Van Slyke, D. D.: The Analysis of Protein by Determination of the Chemical Group Characteristics of the Different Amino Acids, J. Biol. Chem. **10**:15, 1911-1912. (*b*) Koehler, A. E.: Modification of the Van Slyke Method for Determining Arginine, ibid. **42**:267, 1920.

12. Van Slyke, D. D.: Improvements in the Method for Analysis of Proteins by Determination of Chemical Group Characteristics of the Different Amino Acids, J. Biol. Chem. **22**:281, 1915.

TABLE 1.—*Elementary Composition of Proteins* *

Preparation	Nitrogen, per Cent*	Carbon, per Cent*	Hydrogen, per Cent*	Sulphur, per Cent*	Ash, per Cent
Albuminoid	16.65	52.42	7.17	0.70	0.946
Albuminoid	16.69	52.37	7.18	0.69	0.903
Average	16.67	52.40	7.18	0.70	0.925
Alpha crystallin	16.69	52.17	7.18	0.61	0.436
Alpha crystallin	16.64	52.22	7.18	0.605	0.381
Average	16.66	52.20	7.18	0.608	0.409

* The percentages were calculated on an ash-free and moisture-free basis. Carbon and hydrogen were estimated by Pregl's method.

TABLE 2.—*Distribution of Nitrogen in Albuminoid as Determined by the Van Slyke Method* *

Nitrogen	Preparation				Average, per Cent
	I,† Gm.	II,‡ Gm.	I, per Cent	II, per Cent	
Total	0.4001	0.4196	16.40	16.43	16.41
Amide	0.0034	0.0029	0.85	0.69	0.77
Humin	0.0039	0.0042	0.97	1.00	0.98
Cystine	0.0049	0.0052	1.22	1.23	1.23
Arginine	0.0769	0.0788	19.21	18.83	19.02
Histidine	0.0193	0.0262	5.82	6.23	6.02
Lysine	0.0251	0.0286	6.25	6.70	6.47
Amino of filtrate	0.2086	0.2187	52.13	52.05	52.09
Nonamino of filtrate	0.0557	0.0561	14.00	13.60	13.80
Recovered	0.3981	0.4206	99.45	100.85	

* The figures for nitrogen are corrected for the solubilities of the phosphotungstates of the basic amino-acids.
† 2.4391 Gm. of protein.
‡ 2.5538 Gm. of protein.

TABLE 3.—*Distribution of Nitrogen in Alpha Crystallin as Determined by the Van Slyke Method* *

Nitrogen	Preparation				Average, per Cent
	I,† Gm.	II,‡ Gm.	I, per Cent	II, per Cent	
Total	0.4509	0.3234	16.77	16.66	16.71
Amide	0.0076	0.0055	1.70	1.70	1.70
Total humin	0.0098	0.0073	2.17	2.25	2.21
Cystine	0.0073	0.0051	1.61	1.57	1.59
Arginine	0.0977	0.0705	21.66	22.10	21.88
Histidine	0.0237	0.0213	5.25	6.58	5.91
Lysine	0.0112	0.0064	2.48	1.97	2.22
Amino of filtrate	0.2307	0.1655	51.16	51.20	51.18
Nonamino of filtrate	0.0590	0.0426	13.08	13.19	13.13
Recovered	0.4470	0.3246	99.11	100.56	

* The figures for nitrogen are corrected for the solubilities of the phosphotungstates of the basic acids.
† 2.6884 Gm. of protein.
‡ 1.9469 Gm. of protein.

TABLE 4.—*Distribution of Nitrogen in Albuminoid and Alpha Crystallin as Calculated from the Van Slyke Analyses in Terms of Percentage of the Proteins*

Nitrogen	Albuminoid			Alpha Crystallin		
	I, per Cent	II, per Cent	Average, per Cent	I, per Cent	II, per Cent	Average, per Cent
Amide	0.13	0.11	0.12	0.28	0.28	0.28
Humin	0.16	0.16	0.16	0.36	0.37	0.36
Basic	5.32	5.59	5.45	5.41	5.35	5.38
Nonbasic	10.69	10.60	10.64	10.77	10.68	10.73
Total	16.30	16.46		16.80	16.68	

DETERMINATION OF TYROSINE TRYPTOPHAN AND CYSTINE

The determination of tyrosine and tryptophan was done according to the colorimetric method of Folin and Marenzi.[13] In addition to the values found for cystine by the Van Slyke method, there are also included in table 5 figures for this amino-acid obtained colorimetrically by the Sullivan method.[14] Since the Van Slyke method is known to give uncertain results for cystine and the Sullivan method is based on a high degree of specificity, the percentages obtained by the latter method are to be regarded as expressing more accurately the actual amount of cystine present in the proteins. There is no accurate method known for the estimation of cystine and cysteine as separate amino-acids in protein.

TABLE 5.—*Basic Amino-Acids in Albuminoid and Alpha Crystallin* *

Amino-Acid	Protein	
	Albuminoid, Average per Cent	Alpha Crystallin, Average per Cent
Arginine (percentages calculated from the average results of duplicate analyses by the Van Slyke method)	9.91	11.24
Histidine (percentages calculated from the average results of duplicate analyses by the Van Slyke method)	3.30	3.65
Lysine (percentages calculated from the average results of duplicate analyses by the Van Slyke method)	5.62	1.87
Cystine (percentages calculated as stated. Not to be considered as actual cystine, but as an arbitrary figure)	2.63	2.36
Cystine (determined by the Sullivan colorimetric method)	1.25	1.11
Tryptophane (determined by colorimetric method of Folin and Marenzi)	0.713	0.85
Tyrosine (determined by colorimetric method of Folin and Marenzi)	6.25	5.31

* Calculated on an ash-free and water-free protein.

COMMENT

The method of Mörner [9] was used at first for the preparation of the proteins. Without careful control of the p_H of the solutions, the proteins that were isolated were found to contain small amounts of the other soluble proteins in spite of repeated precipitations. The pure albumin and beta crystallin could not be isolated by Mörner's method. The small amounts of the contaminating proteins, as Jess believes, would probably make only a slight difference in many of the analyses, but in specific analyses of beta crystallin and albumin, which are difficult to purify, the difference would be noticeable. Jess isolated the individual amino-acids from the proteins in an excellent manner with the method that he used.

13. Folin, O., and Marenzi, A. D.: Tyrosine and Tryptophane Determinations in One-Tenth Gram of Protein, J. Biol. Chem. **83**:89, 1929.

14. Sullivan, M. X.: A Distinctive Test for Cystine, Pub. Health Rep. **41**: 1030, 1926; The Colorimetric Estimation on Cystine in Casein by Means of the Naphthoquinone Cysteine Reaction, J. Biol. Chem. (Proc. Soc. Biol. Chem.) **74**:xiv, 1927.

Since albuminoid is insoluble in salt solutions it cannot be classified as an euglobulin, and yet it cannot be considered as an ordinary albuminoid such as elastin, collagen or keratin. It may be a modification of a globulin, since the nitrogen distribution approximates that of alpha-crystallin, but it cannot be called a true or a pseudoglobulin on the basis of its physical characteristics. The analysis of albuminoid is yet too incomplete to permit classification. There is evidence that there may be more than one albuminoid.

Alpha crystallin behaved in dialysis like pseudoglobulin of the blood, and beta crystallin like euglobulin. However, the two globulins of the blood are practically identical, but the alpha and beta are entirely different chemically, as shown by analyses. It is not known whether or not beta crystallin is precipitated with lecithin in the same manner as euglobulin.[10b] According to the data of Jess, no glycine was found. This also suggests that these two proteins are different from globulins and correspond to the animal albumins.

The p_H of the solutions, which show minimal solubility of the proteins, are not to be considered as the true iso-electric zones, because it is well known that the salt effect and temperature may change the zone considerably. The elementary analyses of the proteins are practically the same as previously reported by Jess and Mörner.

The analyses according to the method of Van Slyke and the colorimetric methods show some variations from qualitative data previously published. It was to be expected that the amount of tyrosine was higher than Jess found. The analyses for other amino-acids agreed well with the values found by the qualitative isolation method.[15] Without a doubt the amount of cystine and cysteine obtained by Jess as cystine is entirely too high. The sulphur content of the proteins cannot be considered as derived entirely from cystine. The method as used by Jess [10a] has been proved by other investigators to destroy a part of the cystine. At the present time the method of Sullivan perhaps gives the nearest estimation of the actual amount of this amino-acid. The method of Folin and Marenzi [16] was not applied successfully with various amounts of protein. The amount of tryptophan was estimated for the first time. Methionine, ergothioneine and other sulphur compounds are yet to be isolated from these proteins. A mucoprotein containing sulphur as a sulphate may be also expected to be present.

The determination of cystine in whole lens has a questionable value in itself. The results obtained by Tassman and Karr [17] may be criti-

15. Jess, A.: Der Gehalt der Linsenproteine an Histidin, Arginin und Lysin, Ztschr. f. physiol. Chem. **122**:160, 1922.

16. Folin, O., and Marenzi, A. D.: An Improved Colorimetric Method for the Determination of Cystine in Proteins, J. Biol. Chem. **83**:103, 1929.

17. Tassman, I. S., and Karr, W. G.: Glutathione in the Crystalline Lens, Arch. Ophth. **2**:431 (Oct.) 1929.

cized. The total cystine, according to their method, means that both the cystine and the cysteine were determined together as cystine. The cysteine of glutathione was calculated as the total amount of cysteine. If a glutathione is considered as a tripeptide,[18] glutamyl cysteinyl glycine, instead of the dipeptide, glutamyl cysteine, the amount of cysteine must be less. However, this cysteine is not free, but is a part of glutathione. The possibility of insoluble cysteine of the protein molecule was disregarded. Unfortunately, there is no method of analysis for the determination of the cysteine or cystine of the protein molecule as separate amino-acids. The test by Folin and Looney [19] is not regarded specific for cystine [14] by many investigators.

The nitroprusside test must be carefully considered before accepting the results obtained from lenses. It must not be forgotten that acetone, aceto-acetic acid and other compounds free from sulphur and other disulphides give this test. The test indicates the presence of free cysteine, reduced glutathione or a sulphydryl group, but does not distinguish between them. Abderhalden and Wertheimer [20] considered that the free cysteine or bound cysteine was in the tissue in other ways than as glutathione. Tunnicliffe [21] and Hopkins believed that the soluble sulphydryl groups in the tissues belong to glutathione and there is no evidence of the existence of free cysteine in normal tissue. The question of the occurrence of free cysteine and cystine in normal tissue or lens, however, is still open.

CONCLUSION

An improved method for the preparation of albuminoid and alpha crystallin was developed. The quantitative analyses of albuminoid and alpha crystallin was made. The amount of cystine (cystine and cysteine) was found to be lower than recorded in previous reports. The comparison of the analyses of the albuminoid and alpha crystallin of the lens indicates that they are dissimilar.

The material was obtained through Mr. R. L. Fox. The technical procedures were performed with the aid of Mr. W. Tauber.

18. Hopkins, F. G.: On Glutathione: A Reinvestigation, J. Biol. Chem. **84:** 269, 1929. Kendall, E. C.; Mason, H. L., and McKenzie, B. F.: A Study of Glutathione: IV. Determination of the Structure of Glutathione, J. Biol. Chem. **88:**409, 1930.

19. Folin, O., and Looney, J. M.: Colorimetric Methods for the Separate Determination of Tyrosine, Tryptophane and Cystine in Proteins, J. Biol. Chem. **51:**421, 1922.

20. Abderhalden, E., and Wertheimer, E.: Studium über Autoxydation: II. Versuche über die Umwandlung vom Cystein im Cystin unter verschiedenen Bedingungen, Arch. f. d. ges. Physiol. **198:**122, 1923.

21. Tunnicliffe, H. E.: Glutathione: The Occurrence and Quantitative Estimation of Glutathione in Tissues, Biochem. J. **19:**194, 1925.

PATHOGENESIS OF RETINITIS PIGMENTOSA

WITH A NOTE ON THE PHAGOCYTIC ACTIVITY OF MÜLLER'S FIBERS

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AND
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In the past, studies on the pathogenesis of retinitis pigmentosa have been concerned mainly with efforts to discover the remote causes of the disease: the influence of heredity and of consanguinity between the parents, the possible disturbance of liver function, the possible relation of vitamin deficiency, the relation of the ocular disturbance to associated conditions in other organs such as deafness, endocrine disturbances, mental defect, epilepsy, etc. The aim of the present investigation is much more modest, and is concerned with an attempt to unravel the local manifestations of the disease, and to discover what aspect of the local lesion may be considered primary and what aspects may be considered secondary to this primary lesion. For example, the disease process manifests itself in the death and destruction of the retinal neuro-epithelium, and of the retinal ganglion cells. One of the questions that we wish to inquire into is whether both these cell layers are directly affected by the same remote, and as yet unknown, influence, or whether the changes in one group of cells are of such a character as to cause directly a destruction of the other group of cells.

Our knowledge of the histopathology of retinitis pigmentosa has recently been enhanced by the studies of Verhoeff [1] and of Koyanagi.[2] In summary, the lesions found by these authors and by the older classic writers consist in a disturbance in the retinal pigment epithelium with a scattering of pigment granules in the retina. The granules are in large part picked up by wandering cells which carry them to the perivascular spaces about the retinal capillaries and venules, but numerous isolated pigment granules may be found elsewhere in the retina. There are atrophy and destruction of the retinal neuro-epithelium, the rods and cones and their cell bodies. In lesser degree, there are

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From the Wilmer Ophthalmological Institute of the Johns Hopkins University and Hospital.

Read before the Section on Ophthalmology at the Eighty-Third Annual Session of the American Medical Association, New Orleans, May 13, 1932.

1. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **28**:176, 1930.
2. Koyanagi, Y.: Arch. f. Ophth. **127**:1, 1931.

atrophy and disappearance of the retinal ganglion cells and of their fibers in the optic nerve. A marked proliferation of glia has been found, both in the retina and in sheets of tissue spread over its surfaces. Verhoeff has shown that the proliferated glia are derived mainly from Müller's fibers. Finally there are obliterative changes in the retinal blood vessels which, most authors agree, are secondary to the atrophy of the retinal neurons.

The fact that the symptom of night-blindness generally precedes the obvious deposit of pigment in the retina, and that the deposit of pigment generally precedes clinical evidence of optic atrophy has led most authors to believe that the disease of the retinal neuro-epithelium is primary, and that of the other retinal elements secondary. The present investigation consists in an attempt to test this hypothesis experimentally and, in particular, to discover what changes may be expected to take place in the retina as the result of the mere presence in this tissue of granules of melanin pigment. One of us (J. S. F.)[3] has previously reported the discovery, in cases of pigmentary degeneration of the retina occurring as a complication of late neurosyphilis, that vigorous antisyphilitic treatment may not only stop the progress of the lesion, but may also lead to a gradual disappearance of the pigment from the retina. This observation suggested the possibility that, in ordinary cases of retinitis pigmentosa, a continuous supply of pigment to the retina may take place.

The experimental procedure consisted in the injection into the vitreous of the eyes of albino rabbits of a suspension of melanin granules obtained from the pigment epithelium of pigs' or cows' eyes. The pigment suspension was prepared as follows:

Pigs' or cows' eyes in lots of fifty, obtained fresh from the slaughter house, were dipped in boiling water for fifteen seconds, rapidly cooled, and then bisected equatorially under sterile precautions. The retina and vitreous were then removed from the posterior sector with a small forceps, and the retinal pigment epithelium gently scraped off from the surface of the choroid without penetrating Bruch's membrane. The scrapings were suspended in sterile distilled water, and the pigment recovered by centrifugation. In order to break up the epithelial cells and release the melanin granules, the sediment was transferred to a sterile mortar and mixed with dry crystals of sodium chloride. The resulting paste was dried in a vacuum and then ground to a fine powder. A few drops of sterile distilled water were added, and the grinding continued until smears of the mixture showed the melanin granules to be isolated from one another without any clumps. Sufficient water was added to dissolve the sodium chloride. The pigment was then recovered, washed by repeated suspension in distilled water and repeated centrifugation at high speed, and finally suspended in 40 cc. of physiologic solution of sodium chloride containing 0.5 per cent cresol, U.S.P. The suspension was placed in an incubator for forty-eight hours, after which a culture was taken, which proved to be sterile. The suspension, which had a dark brown color, was now ready for use and was preserved in the refrigerator at 6 C.

3. Friedenwald, J. S.: Am. J. Ophth. **13**:943, 1930.

Two-tenths cubic centimeter of the pigment suspension was injected into the vitreous of one eye of each of twelve albino rabbits, the injection being made at the equator of the eyeball behind the insertion of the superior rectus muscle. As a control an equal volume of 0.5 per cent cresol in physiologic solution of sodium chloride was injected into the opposite eye of each rabbit. The animals were observed for periods varying from four to eighteen months and were then killed. Their eyes were fixed in Bouin's solution and sectioned for histologic study. The control eyes showed no reaction to the injection, and on section showed no lesions except the scar of the puncture hole. The eyes receiving the injection of pigment all exhibited such uniformity in their subsequent course that we can describe them as a group.

OPHTHALMOSCOPIC APPEARANCE

The pigment emulsion, which immediately after the injection could be seen as a circumscribed black mass in the upper part of the vitreous, gradually, in the course of several weeks, sank to the lower periphery of the vitreous leaving the upper portion clear. Slowly the sharp outlines of the mass became broken up, and small detached black globules became visible. These on section proved to be agglutinated masses of phagocytic cells packed with pigment. The transport of the pigment out of the vitreous was extremely slow, and even in the longest surviving animals was never complete. No pigment appeared at any time in the anterior chamber. Between one and two months after the injection, deposits of pigment in and on the retina in the lower periphery could be recognized. These consisted in irregular black masses, sometimes fairly circular in form, but often linear or branching. In addition to these larger masses, a fine peppering of the retina in this region was noted. Occasionally masses of pigment were seen in the vascular part of the retina near the disk. Here the pigment generally surrounded the blood vessels, and exhibited the familiar bone marrow pattern. In the course of time, these pigment masses near the disk were seen to break up and disappear, and at the end of the experiment in the animals which survived longest, it was obvious that a good deal of the pigment had been removed from the eyes. In the oldest lesions a grayish opacity and streaking of the retina were noted.

MICROSCOPIC APPPEARANCE

On section large clumped masses of phagocytes (monocytes) packed with pigment were found in the vitreous. Similar masses were found resting on the retina, and, in the neighborhood of these masses, numerous isolated wandering cells were to be seen in the retina, mainly in the layers nearest the vitreous. In those eyes which were enucleated early

in the series (from four to six months after the injection of pigment) many Müller's fibers were found packed with pigment granules (fig. 1). With the oil immersion objective, it could be seen that the granules were actually within the cell body of the Müller's fibers, and not merely deposited on the surface. The granules were not, however, limited to the plump part of the cell body near the nucleus, but in most instances were spread along the brushwork of fibers, as far down as the external limiting membrane, and up to the inner surface of the retina. Within the brushwork, the rod-shaped granules of pigment all tended to arrange themselves in the same direction, perpendicular to the plane of the retina.

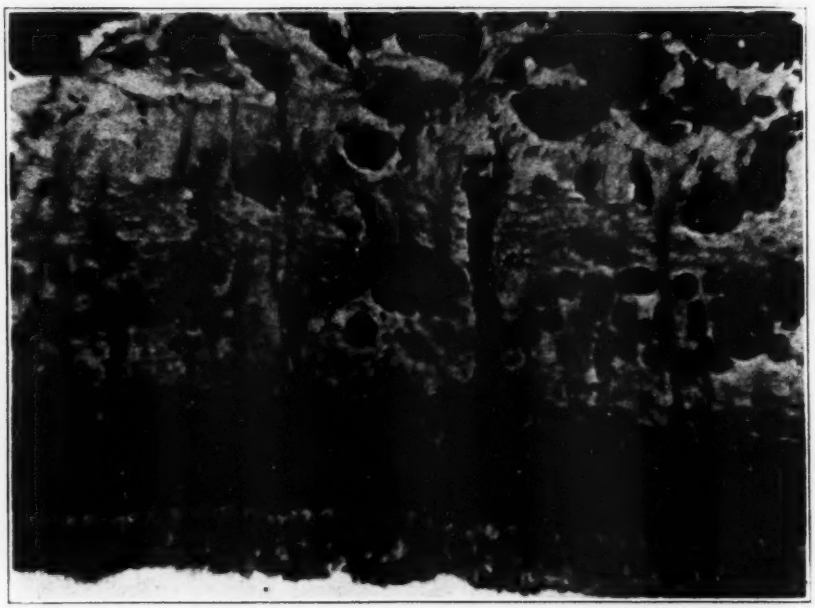

Fig. 1.—Phagocytosis of melanin granules by Müller's fibers.

In eyes enucleated at longer periods after the injection, phagocytosis by Müller's fibers could still be seen, but was somewhat obscured by the marked proliferation of these same fibers, which in many places formed a thick layer of tissue on the surface of the retina (fig. 2). In these later stages considerable destruction of retinal tissue could be found. Under some of the larger deposits of pigment, the retina was completely disorganized, none of the three layers of neurons could be recognized, and only a thin glial sheet remained, heavily infiltrated with monocytic phagocytes containing pigment (fig. 3). At the edge of such a lesion, the layer of bipolar cells was usually the first to be found presenting a normal appearance. The extent of damage to the neuroepithelium was therefore greater than that to the bipolar cells in spite

of the fact that the pigment entered the retina from the vitreous side. The extent of damage to the ganglion cells was greater than that to the neuro-epithelium. In one eye the degeneration of the retina at one spot was so extensive that some phagocytes penetrated to its outer surface. Along with these were found scattered pigment granules mainly deposited on the surface of the (in the albino) otherwise unpigmented cells of the pigment epithelial layer.

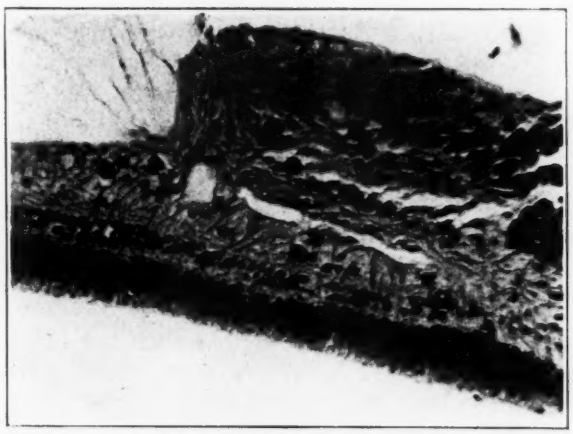

Fig. 2.—Proliferation of Müller's fibers.

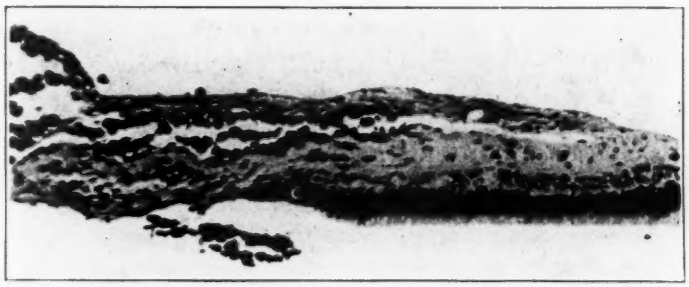

Fig. 3.—Phagocytes in the retina with disorganization of the retinal structure and disappearance of the neurons.

In addition to the pigment contained in monocytic phagocytes and in Müller's fibers, large numbers of granules were found scattered through the retina, isolated and in clumps. It was impossible to tell in regard to this portion of the pigment whether the granules were actually phagocytosed by and contained in the retinal cells, or whether they were lying on surfaces of cells. The retinal cells in such regions showed no evidence of damage.

COMMENT

The results of these experiments raise two sets of problems: (1) the interpretation of the observations in the experimental animals, and (2) the application of these observations to the problem of the pathogenesis of retinitis pigmentosa.

1. It seems clear from the histologic study of these rabbits' eyes that the pigment granules are completely inert and passive. In those regions in the retina where phagocytes have not penetrated, the granules rest in or among the retinal cells without causing the slightest reaction. The granules, even those contained in phagocytes, maintain their bacillary form unchanged for as long as eighteen months. It is doubtful whether the cells of the host can even digest these granules. It must be noted that in each animal only one injection of pigment was given, so that no opportunity was afforded for the host to become hypersensitive to any soluble proteins which may have adhered to the granules in spite of the care in their preparation. If the granules are completely inert and undigestible, the only possible explanation for the damage and disorganization found in the retina is that this had resulted from the infiltration with phagocytes. A structure so highly and so delicately organized as the retina may, therefore, be supposed to suffer disorganization when an army of heavily laden phagocytes tramp through. Such, at least, is the impression that one gains from studying these sections and noting that the disorganization of the retina is present only where large numbers of phagocytes were present.

Phagocytic activity of Müller's fiber cells has not, so far as we know, been observed previously, though phagocytosis by other retinal glia has been reported. Whenever glia cells are found to be phagocytic, the question may legitimately be raised as to whether they are truly epithelial in origin, and whether they may not be related to the microglia of Hortega. In regard to Müller's fibers, this can definitely be answered in the negative. According to Mann,[4] Müller's fibers appear in the earliest differentiation of the retinal cells, before the primitive retina is invaded by mesenchyma.

2. Proliferation of Müller's fibers is seen in other conditions beside retinitis pigmentosa and in the animal experiments that we have reported. What is of interest in this regard is that the presence of melanin granules in the retina is in itself an adequate stimulus to this proliferation. No other more remote cause need be sought to explain this observation in retinitis pigmentosa. The same conclusions may be drawn in regard to the death of the retinal neurons. The experiments demonstrate that the retinal ganglion cells can be destroyed as the direct result of the presence

4. Mann, Ida C.: The Development of the Human Eye, London, Cambridge University Press, 1928.

in the retina of phagocytes filled with melanin granules. It must be remembered that in typical cases of retinitis pigmentosa the heavy traffic of pigment through the retina continues for many years. From what we have found in our rabbits, the cumulative effect of such long-continued infiltration with pigment and phagocytes must be considerable.

This aspect of the problem raises the question as to whether or not atrophy of the retinal ganglion cells may occur in cases of retinitis pigmentosa sine pigmento. Up to the present no such case has been examined histologically. We have not been able to find reported a single case of definite optic atrophy in the clinical reports of such cases. It would appear that the degree of atrophy of the optic nerve is closely correlated with the amount and duration of the pigment in the retina. It must be remembered, however, that there is a great deal more pigment scattered in the retina in cases of retinitis pigmentosa than is visible with the ophthalmoscope. In the periphery of the fundus, the definition of the ophthalmoscopic picture is such that objects less than from 30 to 50 microns in diameter cannot be distinguished. This means that the smallest black dot visible in the periphery of the retina consists in a clump of half a dozen or more phagocytes. Yet, in sections, one finds great numbers of isolated phagocytes laden with pigment granules and, in addition, great numbers of granules apparently free in the retina. It seems likely, therefore, that many cases that appear clinically as "sine pigmento" are not actually without pigment in the retina.

From our experiments, we conclude that no primary retinal lesion outside of the neuro-epithelium is necessary to account for the clinical and histologic pictures of this disease. The experiments do not enable us to decide whether in the natural disease both elements of the neuro-epithelium are primarily involved, or whether the disintegration of the rods and cones is secondary to the scattering of pigment from the pigment epithelium or vice versa.

CONCLUSIONS

1. The presence of melanin granules in the retina causes a destruction of the retinal neurons and a proliferation of Müller's fiber cells. The retinal ganglion cells and the rods and cones seem to be more easily injured by this agency than the bipolar cells. The direct cause of the injury seems to be the disorganization of the retinal structure produced by the migration through the retina of wandering cells laden with pigment granules.

2. All the histologic changes in the retina in retinitis pigmentosa can be accounted for on the hypothesis of a primary lesion to the retinal neuro-epithelium with secondary long-continued infiltration of the retina by melanin granules.

3. Müller's fiber cells are capable of active phagocytosis.

ABSTRACT OF DISCUSSION

DR. F. HERBERT HAESSLER, Milwaukee: This highly entertaining experimental study has shed important light on the biology of some of the constituent cells of the retina. Müller's fibers are phagocytic for pigment granules. This fact, while not startling *a priori*, is important because it is established for the first time that glia cells of epithelial origin are phagocytic. It is generally believed among neuropathologists that only glia cells of mesodermal origin have phagocytic function.

The behavior of the pigment is interesting. The pigment itself, no matter how introduced into the retina, whether from the exterior or the internal surface, is able to destroy the rods and cones in a pattern which is characteristic of retinitis pigmentosa. The actual arrangement of the pigment throughout the retina has no particular significance aside from the fact that pigment which has entered the retina is caused to leave it. If, as a matter of speculation, the cells which are loaded with pigment reach the liver, it is possible that the hepatic function will be interfered with in cases of long-continued irritation of the retinal pigment. This, of course, has not been established by this piece of work or by any other, but it has been shown that there is a relationship between hepatic function and retinitis pigmentosa. Of course, the relations between the eye and the liver are manifold, and it is perhaps within the realm of possibility that long-continued deposition of pigment in the liver may be caused by interference with its function from retinitis pigmentosa.

DR. PARKER HEATH, Detroit: This paper is of considerable histologic interest. The experimental evidence of the influence of secondary inflammatory reactions on the retina is well demonstrated. The same phenomena take place without pigment and without the overaction of Müller's fibers in many other retinal lesions —such as circinate retinitis and the chemical and cellular reactions of the aging process.

In general, clinical and experimental evidence has well established that the main destroying factor is an overwhelming defensive response.

Dr. Friedenwald has experimentally demonstrated the storage of pigment tissue extract in Müller's cells. In retinitis pigmentosa sine pigmento, although not studied microscopically, some agent other than pigment, such as one or a group of lipids, could initiate the process and produce the clinical picture. Experimental studies along these lines should be undertaken.

I should like to ask Dr. Friedenwald whether he thinks that in spite of the single injection of pigment substance any of the cellular reactions in his experiments resembled the so-called anaphylactic response.

DR. S. B. MUNCASTER, Washington, D. C.: I wish to give my experience with sodium cacodylate in the treatment of retinitis pigmentosa by a short report of two cases.

CASE 1.—A school teacher, aged 55, came to my office in 1906, complaining of blurring of vision. On examination I found that she had retinitis pigmentosa in each eye. Vision in the right eye was 20/200 — 1.50 at 90 20/40-2; that in the left eye, 5/200. There was no improvement with glasses. I prescribed a capsule containing one-half grain (0.03 Gm.) of sodium cacodylate twice daily and entire rest for the eyes for three months. The vision improved so that she was able to return to work and continue until she was retired. I examined her eyes last week and found the field of vision contracted about one-third. This patient is now 81 years of age. Nearly all the bone cell pigment deposits have

disappeared, and atrophic changes in the blood vessels and retina have taken place. She can see enough to read for one or two hours a day. This patient has been taking capsules of sodium cacodylate for twenty-six years. After taking them constantly for three years, she would stop for a month and sometimes longer.

CASE 2.—A girl, aged 8 years, was brought to my office in 1928. On examination I found that she had retinitis pigmentosa. Vision in the right eye was 8/200; that in the left eye, 20/200. There was no improvement with glasses. She was given a capsule containing one-fourth grain (0.01 Gm.) of sodium cacodylate twice daily. Her eyes improved in eight months, the right eye from 8/200 to 20/100, and the left eye from 20/200 to 20/40. By forcing her eyes, she can see ordinary print. She came to my office one morning as her eyes were not good. I asked her what she had been doing. She said that she had written a play for some girls all the day before and in the evening. I had her parents send her to a school to learn the Braille system, and she can now read and write Braille. I feel confident that the continuous use of sodium cacodylate will save the sight of this patient. She has been taking capsules of sodium cacodylate, which are made fresh every month. Sometimes she stops taking the medicine for two weeks. She has a peculiar field of vision all around the periphery, the streaks of vision extending down, forming a V from the upper field.

The capsules have to be made up every month; otherwise, they do not accomplish what we want. I have other patients with retinitis pigmentosa, but have not been able to follow the result of the treatment. These two patients have night blindness. The blood is normal.

DR. JONAS FRIEDENWALD: Dr. Haessler has raised an interesting speculative point as to whether or not the associated hepatic disturbance, which is found in some cases of retinitis pigmentosa, may be due to damage to the liver by the pigment from the eye. Up to the present most people who have speculated about the relation between disturbance of the liver and ocular disease have been inclined to place the hepatic disturbance first and the ocular lesion second. So far as we know, there is no chemical difference between the pigment in retinitis pigmentosa and the melanin in the choroid and skin. In sunburn there is proliferation of the melanin of the skin and when the tan fades the pigment may be destroyed or carried away in far larger quantities than any that could be derived from the eye. Nevertheless, the liver is not damaged by such an event.

In regard to the possible allergic character of the reaction of the retina that we have described, it is, of course, true that the pigment we used was obtained from cows or pigs and was injected into rabbits. It may have carried along with it some soluble protein foreign to the rabbit. However, we were careful to make only one injection into the animal's eyes, and any allergic state that may have developed following the first injection was not stimulated into reaction by a second.

The histologic pictures which I showed yesterday in connection with Dr. Woods' paper showed that the normal nonallergic person's histologic reaction to melanin granules was characterized by an invasion of the pigment deposit by large monocytic cells, while in the allergic patient there were epithelioid and giant cells. The reaction we found in rabbits' eyes in the present experiments, therefore, was similar to that found in nonallergic persons. Our experiment shows that phagocytosis by Müller's fibers takes place only under special conditions in the early stages of the reaction and disappears later on. The absence of this feature in cases of senile macular degeneration might depend on the particular time at which the eye was examined histologically.

HISTOLOGIC CHANGES IN AN EYE EIGHT YEARS AFTER SCLEROCAUTERY PUNCTURE FOR SEPARATION OF THE RETINA

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I have been unable to find in the literature any record of a microscopic examination of a human eye following sclerocautery puncture. It seems well, therefore, that the following case should be put on record.

REPORT OF A CASE

History.—A man, aged 25, sustained a severe contusion of the left eye by a baseball. He immediately noticed visual deterioration, which progressed. In three months the nasal field of vision was lost in the injured eye. Nine months after the injury, in June, 1923, the patient came to the Massachusetts Eye and Ear Infirmary for treatment.

Examination.—Dr. F. H. Verhoeff found a temporal and nasal separation of the retina and two holes in the retina. There were numbers of floating vitreous opacities. The right eye was normal.

Operation and Course.—The patient was operated on by Dr. Verhoeff, who reported as follows: "A conjunctival flap was made down to the external rectus muscle. The muscle fibers were split longitudinally until the bare sclera was revealed. At a point about 12 mm. from the limbus temporally beneath the muscle, a hole was burned in the sclera [and choroid] with a ball tip galvanocautery. There was an escape of subretinal fluid. The conjunctival wound was sutured. Then an incision with a cataract knife was made about 12 mm. from the limbus nasally through the internal rectus muscle into the globe at the front of the medial retinal separation. A small amount of retinal fluid escaped."

The following day no separation of the retina was seen. On the second day a small separation was seen just below the location of the retinal holes. This separation increased daily. Five days after the first operation, a second sclerocautery puncture was done 12 mm. from the limbus down and temporally. There was a small escape of fluid, and the vitreous presented but it did not escape.

The retina was again in place for two days, but on the third day the separation recurred and a third cautery puncture was done, down and nasally. The separation recurred after two days, and the patient was discharged with the comment that the condition was hopeless.

These operations were done in 1923, before it was the general practice to attempt to "close" the holes in the retina.

The patient was seen after a lapse of eight years (in October, 1931). He gave a history of severe pain and inflammation in the left eye for three days. Vision was nil, the cornea steamy, the anterior chamber shallow, the iris discolored and

From the Massachusetts Eye and Ear Infirmary.

the tension 60 mm. with the Schiötz tonometer. The fundus could not be seen. The right eye was normal.

The left eye was enucleated. After fixation in a diluted solution of formaldehyde, U.S.P. (1:10) for forty-eight hours and after immersion in an 80 per cent solution of alcohol containing 2.5 per cent hydrochloric acid for twenty-four hours, the eye was opened. After being embedded in celloidin, microscopic sections were made.

Macroscopic Observations.—The retina was completely separated by a slightly flocculent fluid, except at the margin of the disk, at the ora serrata and at the site of the three cautery punctures. The disk was deeply cupped. Several large cysts were seen in the separated retina, but no holes were seen. As the retina was separated, it cut very irregularly, and it is possible that the holes seen at the first admission were cut across when the eye was opened and therefore were not found.

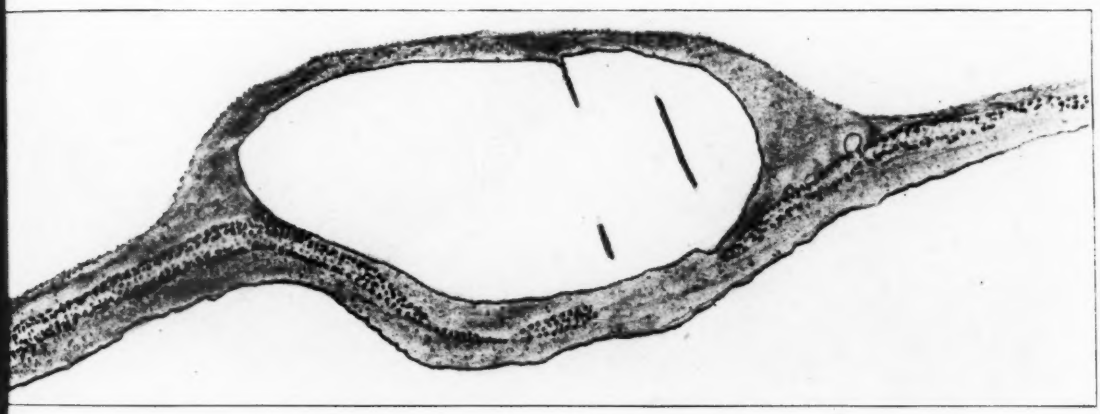

Fig. 1.—A large cystoid space in the retina containing strands of retinal tissue that bridge across the space.

Microscopic Observations.—Early bullous keratitis, sclerosis of the ligamentum pectinatum and deep glaucomatous cupping of the optic disk were present. The vitreous took a deeper stain than usual. A delicate fibrillar structure could be seen throughout the vitreous. The retina contained numerous cystoid spaces, some of which were particularly large, being 2 mm. long and 1 mm. wide. Most of the spaces were apparently empty, but some spaces contained a fluid of sufficient albuminoid content to have formed a coagulum. Samuels [1] considered this indicative of recent formation. Some of the large cysts contained a few fine strands of retinal tissue bridging across the space (fig. 1). This suggests strongly that the large cysts were formed by coalescence of the smaller adjacent cysts. Samuels [1] stated that cysts of the retina are most frequently encountered in detached retina, and in this case I think that the cystoid changes in the retina were largely secondary to the separation.

In sections through the first sclerocautery puncture (fig. 2), where vitreous did not present at the time of operation, the retina was completely disorganized

1. Samuels, Bernard: Cystic Degeneration of the Retina, Tr. Am. Ophth. Soc. **28**:169, 1930.

and reduced to a thin layer of neuroglia. This layer of neuroglia was fused with a mass of scar tissue, replacing the sclera and choroid. In the scar tissue were irregular nodules of pigment derived from the choroid.

Sections through the second sclerocautery puncture (fig. 3), where vitreous presented at the time of operation, showed a similar picture, with a projection of partially hyalinized scar tissue into the vitreous chamber for a distance of 1.5 mm. This projection of scar tissue ended in processes of variable size similar to a paint brush on which paint had dried. Attached to these brushlike processes,

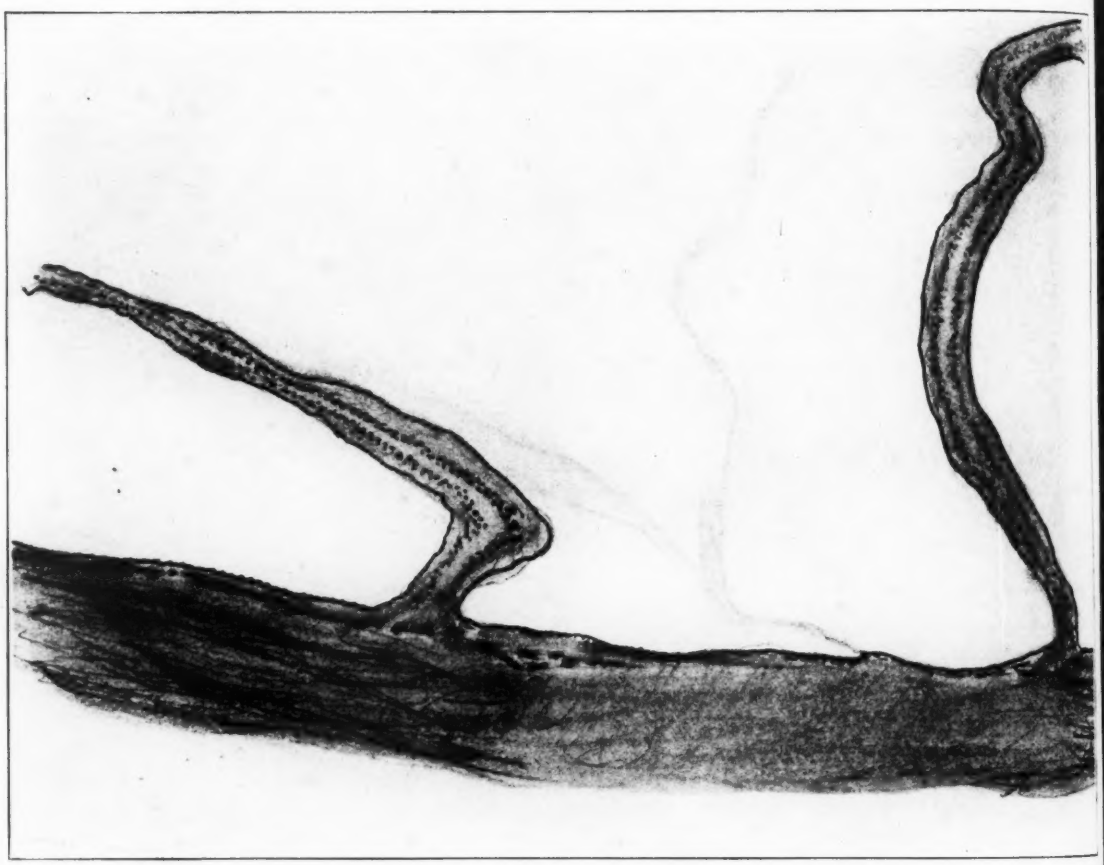

Fig. 2.—Section through the sclerocautery puncture, showing the retina completely disorganized and reduced to a thin layer of neuroglia fused with a mass of scar tissue, which has replaced the choroid and sclera.

and apparently continuous with them, were numerous delicate strands of fibrils that coursed into the vitreous.

Amsler [2] expressed the belief that the success of the operation depends on including vitreous in the scar in order to avoid local trac-

2. Amsler, Marc: Ueber die Gonin'sche Operation, Klin. Monatsbl. f. Augenh. **86**:1 (Jan.) 1931.

tion by the vitreous. At the site of the cautery puncture in my case, where vitreous did not present, the retina was just as adherent as at the site where vitreous did present.

The picture in this case is similar to that obtained by Luntz [3] in laboratory animals.

CONCLUSION

Following sclerocautery puncture, whether vitreous presents at the operative wound or not, the retina becomes firmly adherent to the scar

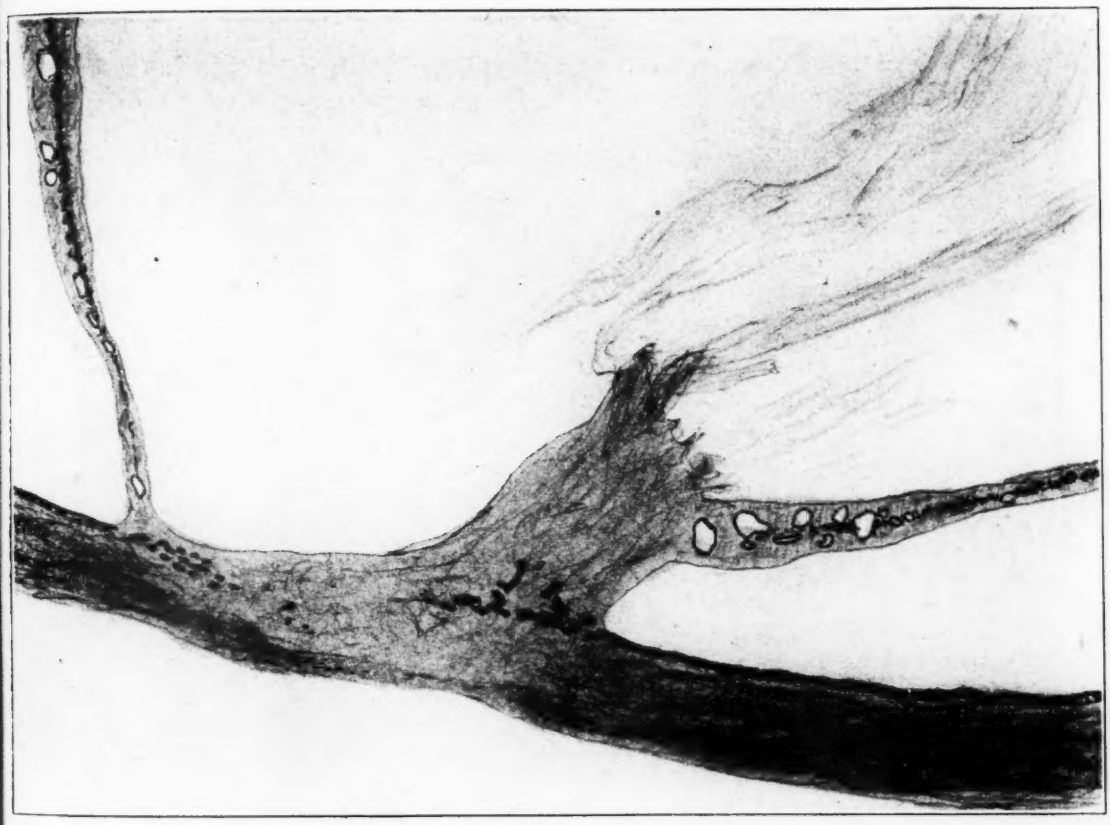

Fig. 3.—Section through the sclerocautery puncture where vitreous presented at the time of operation, showing a mass of partially hyalinized scar tissue projecting into the vitreous chamber from which numerous delicate fibrillar strands extend into the vitreous body.

tissue in the wound. In the case presented, this adhesion was sufficient to withstand the traction exerted by a separated retina for eight years.

128 Newbury Street.

3. Luntz, G.: Ueber die Wirkung der perforierenden Kauterisation am Tierauge, Ztschr. f. Augenh. **73**:380 (March) 1931.

SIGNIFICANCE OF CHOROIDAL HEMORRHAGE

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Choroidal hemorrhage is not uncommon, although the textbooks still give the impression that it is rare and difficult to recognize. Examination with modern equipment has made it possible to diagnose it with more certainty and greater frequency.

Traumatic choroidal hemorrhage may appear in various forms, sometimes obscured by attendant conditions. When it is the only evident sign, the blood is seen as a dark red, granular, rounded mass lying beneath the retinal vessels. In my experience this is most commonly near the disk and always the direct result of a rupture of the choroid. At times the tear is small, and in many cases it is so covered with blood that it does not become visible for several days. This hemorrhage tends to preserve its original boundaries and is absorbed by thinning rather than by dissipation, as is noted in the spontaneous extravasation. A distinctive feature of this and perhaps all choroidal hemorrhages is its slow absorption. I have seen a very thin one take almost three months to disappear. The location and progressive changes are well illustrated by two cases.

TRAUMATIC CHOROIDAL HEMORRHAGE

CASE 1.—L. G., a man, aged 36, was struck in the right eye by a limb of a fallen tree ten days before the picture shown in figure 1, *1* was taken. There were three lacerations of the pupillary margin and considerable hyphemia. The disk was round, with a moderately deep central excavation. The retinal vessels were not noteworthy. There were two choroidal hemorrhages; the larger was ovoid and extended from the disk upward more than 2 disk diameters. The other was almost round and about 1 disk diameter in size.

Four and one-half months later, the blood had entirely absorbed, and the tear in the choroid was evident as a horizontal white streak above the disk (fig. 1, *2*).

CASE 2.—A. H., a woman, aged 30, had a BB shot penetrate her left eye. An almost round, granular, dark red, choroidal hemorrhage extended from the nasal side of the disk a little more than a disk diameter (fig. 1, *3*). This was slightly elevated so that the unaltered retinal vessels curved over it. There were several other extravasations, particularly a large one in the vitreous below the disk.

Four months later, the blood had entirely disappeared and the site of rupture was a very delicate whitish-pink streak, scarcely perceptible in the photograph (fig. 1, *4*). There was widespread choroidal pigmentation in the lower part of the fundus. This is not shown in the photograph.

Read before the Section on Ophthalmology at the Eighty-Third Annual Session of the American Medical Association, New Orleans, May 13, 1932.

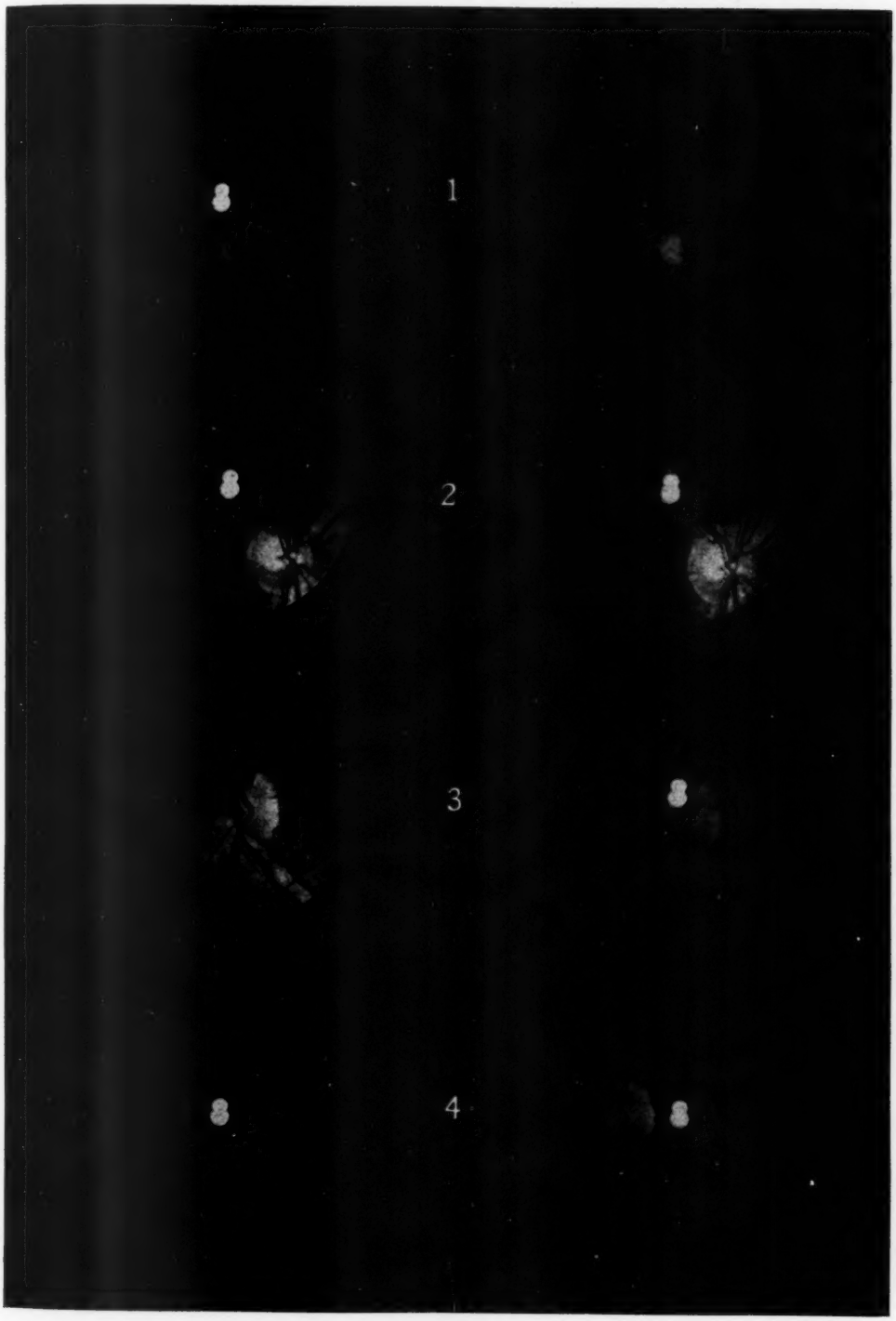

Fig. 1.—Traumatic choroidal hemorrhage (two cases); stereoscopic photographs.

CHOROIDAL HEMORRHAGE FOLLOWING CATARACT EXTRACTION

Choroidal hemorrhage following cataract extraction is not a rarity. The late Dr. S. Lewis Ziegler reviewed the literature up to 1926. His final communication, compiled in his characteristically thorough way, was published in "Contributions to Ophthalmic Science."[1] Lundsgaard, in 1928, referring to his experiences and those of others, stated that it is more to be dreaded than infection.

My personal experience is confined to four cases, all of which ended in the shrinking of the globe, two quite small, the others only partially atrophied.

CASE 3.—A woman, aged 50, presented no unusual symptoms. When the corneal section was completed, there was an expulsive hemorrhage forcing the lens and much of the vitreous out through the wound. A shrunken globe resulted.

CASE 4.—Mrs. H. came under observation in 1927, when she was 63 years old. Hyperpiesia had been diagnosed several years before. The vision was 6/200 in the right eye and 20/100 in the left, with posterior cortical opacities.

In 1930, the vision was so reduced that operation was suggested. After months of treatment, regulated exercise and diet, Mrs. H. entered St. Peter's Hospital with a systolic pressure of 230. After a day there she seemed quiet and calm. Under the usual cocaine anesthesia, a corneal section with conjunctival flap was completed with little bleeding, and a small iridectomy caused no oozing. The lens was grasped with capsule forceps, but the capsule came loose as it engaged in the wound. Expression was completed and the toilet finished. The patient then complained of intense pain; the vitreous spurted out followed by a severe hemorrhage, which continued for days. The clot was cut off and the shrunken globe left in place.

The high blood pressure was not influenced by treatment, but as the vision was reduced to light perception, the patient was readmitted a year later. The other eye was operated on, cocaine anesthesia being used, after the administration of barbital, 1½ grains (0.097 Gm.) three times a day for three days. The section was made at the limbus with no conjunctival flap. The pupillary border of the iris was nicked with the de Wecker scissors. The lens was dislocated with the forceps, the capsule broke loose, and the cataract was easily expressed. The iris was replaced and physostigmine (eserine) instilled. The administration of barbital was continued, and healing was uneventful. The patient's final vision with correction was 20/20 (?).

CASE 5.—R. G., a man, aged 65, had a mature cortical cataract on his left eye and marked anterior and posterior opacities in the right adult nucleus layer. Physically, he seemed in good health for one of sedentary habits; his vessels presented no abnormalities, and his systolic pressure was 130.

He entered St. Peter's Hospital, where, after the usual preparation, the left eye was operated on. A limbus section was made with a large, broad conjunctival flap and a small iridectomy. The lens was brought into the wound before the capsule broke, and was easily expressed. There was no evident intra-ocular bleeding.

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1. Ziegler, S. Lewis: The Problem of Choroidal Hemorrhage in Cataract Extraction, in Contributions to Ophthalmic Science, Menasha, Wis., George Banta Publishing Company, 1926.

The following morning he complained that the dressing was stiff. When the dressing was removed, a large oozing clot was seen over the cornea. In spite of all treatment, this bloody sponge continued to ooze red blood for fourteen days. The end-result was a clear 7.5 mm. cornea and a slightly shrunken globe, but one that was not conspicuously small. Obviously, there is no sight in it.

The fourth experience followed the classic description.

CASE 6.—A woman, aged 72, in excellent health and in fine physical condition, was operated on in St. Peter's Hospital. In the right eye there was a brown, gray, mature cataract, which had developed in two years. Vision was light projection and perception. There was no history or physical evidence of previous ocular disease. The left lens showed opacities of the anterior and posterior adult layer with reduced vision.

With the patient under cocaine anesthesia, a limbus section with a large conjunctival flap was made and a small iridectomy performed. The lens was extracted in capsule with very little manipulation and little pressure. When the dressing was about to be placed, the vitreous presented, followed by a gush of blood. The patient complained of intense pain, and shortly after getting to her room began to vomit. Bleeding and vomiting continued two days. The hemorrhage was so great that the blood literally ran down her face. The end-result was a shrunken globe. This destructive hemorrhage came in a healthy patient with no demonstrable changes in the blood vessel and a systolic pressure of 132.

It is not my intention to dwell on this phase of choroidal hemorrhage, for every one of large experience has had similar disappointments. Lundsgaard, who had one case in 1,200 operations for cataract, ascribed a predisposing rôle to chronic iridocyclitis and glaucoma. My four cases showed no evidence of either of these diseases. There seems to be no proved way to prevent some of these lamentable accidents, even with unusual care and prolonged treatment, although barbital seems to have a quieting effect.

Hemorrhage in my cases was immediate, but delayed bleeding after cataract operation has been recorded; expulsive hemorrhage may occur even several days after any intra-ocular operation.

HEMORRHAGE IN "SENILE MACULAR DEGENERATION"

Cases with such an outcome are fearfully depressing to the operator, but they are fortunately rare as compared with the large group of adults who suddenly lose the central vision of one eye and later that of the other. This disease seems to be an arteriosclerosis of the macular twigs in the choroid, and carefully recorded photographic histories of many cases reveal the life cycle of the lesion. The importance of choroidal hemorrhage is especially significant in this condition, which is called senile macular degeneration, and, as it appears under various disguises, several examples are displayed.

CASE 7.—Mrs. D., aged 56, complained of poor vision. She knew that she had lost the sight of her left eye one and one-half years before my first examination. Her blood pressure was 190 systolic and 86 diastolic, and she had albumin and casts.

Vision in the right eye was 5/200 (fig. 2, *1*). A large, bright red choroidal hemorrhage was partially concealed by the smoky retina. The entire fundus was gray with mottled choroidal channels and small, rather straight retinal vessels.

Three months later, the gray film of edematous retina was larger and the hemorrhage so dissipated as to form a crown with paler lines (fig. 2, *2*).

After two months, the blood ring was much larger, thinner and definitely granular in places (fig. 2, *3*). The central retina looked like a layer of gray wool fibers.

Finally, nine months after onset, the macular region was wrinkled and scarred, and over it lay some scattered spots of pigment (fig. 2, *4*). The absorption of the hemorrhage was complete. There had been no change in vision, and none could be expected.

CASE 8.—Mrs. A. J., aged 67, was first seen on Nov. 11, 1930, when she stated that on October 31 she accidentally rubbed her left eye and found that she could not see out of the right. Examination showed: systolic pressure, 190; nonprotein nitrogen, 36; creatinine, 1.58; albumin or sugar, negative, and the Wassermann reaction, negative. The blood count showed: polymorphonuclears, 48; small mononuclears, 37; large mononuclears, 9; basophils, 1, and eosinophils, 4.

Vision was 1/200. Externally, the eye was normal (fig. 3, *1*). The disk was slightly oval with a small central excavation. The retinal blood vessels were not especially noteworthy in caliber or course. There was a large, dark red patch which included the macula in its upper portion and extended far down below the inferior temporal vein, so that the total area was five times the disk diameter in size. The densest part of the hemorrhage formed the upper and lower portions, whereas the area through the center was less densely infiltrated, and in places the blood had not seeped beneath the retina, thus giving a normal pink reflex. There was one isolated extravasation beneath the extremity of the inferior temporal vein. The macula was decidedly darker than its surroundings. There was a faint gray zone over the clot, and the retinal vessels crossed the hemorrhage.

At the end of three weeks the clot was moth-eaten in appearance (fig. 3, *2*). This was caused by an overlying arc of retinal exudate in the upper, outer part and an absorption of the choroidal blood in other portions. The retinal vessels could be traced.

Two months later, the hemorrhage had broken up into several more or less distinct islands, most of them long and narrow with a definite granular structure (fig. 3, *3*). The macular region was now definitely outlined, its upper edge covered by a portion of a triangular white web, the prolongations of which were crossed by retinal arteries.

The last picture (fig. 3, *4*) was taken ten months after onset. The vision was 20/200, and the systolic pressure was 165. The region where the hemorrhage was thickest was occupied by a gray, filmy, elevated membrane, which suggested a multilocular cyst in appearance. A few peripheral choroidal hemorrhages persisted.

ABSORPTION OF HEMORRHAGE

The patient with choroidal hemorrhage first notices a blur, which, to the average sufferer, is neither distinctive nor sharply delimited. If the fundus can be seen at this earliest stage, it will usually show a dark, granular, elevated red island, often with several smaller ones grouped irregularly about it. Whether large or small, it destroys the function of part or all of the overlying retina. In large hemorrhages the retina becomes smoky gray or even white, just as it does in sudden closure of

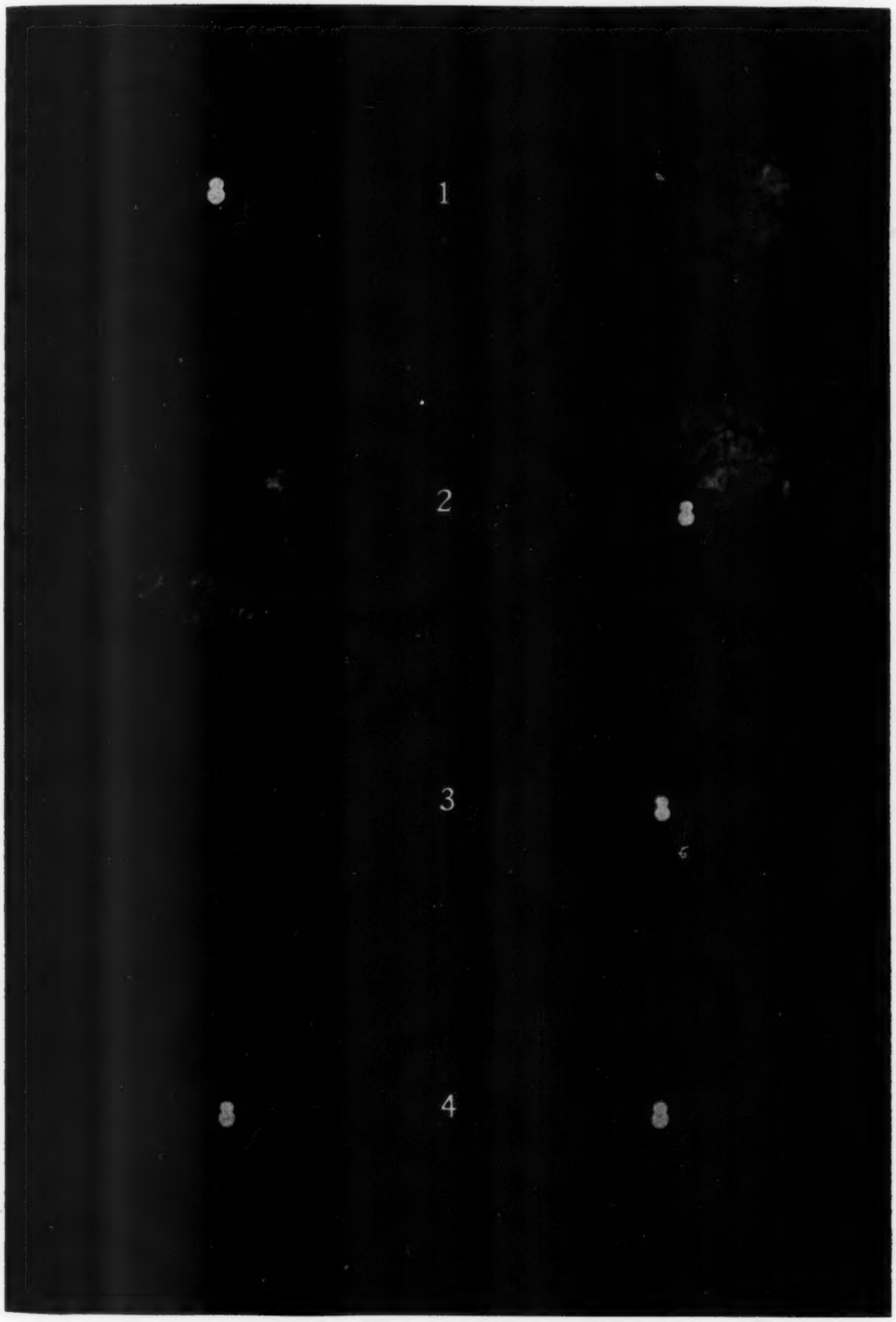

Fig. 2.—Spontaneous macular hemorrhage

Fig. 3.—Choroidal hemorrhage with hypertension.

the central artery of the retina. This delicate shading was aptly likened by Lawson to, "the bloom on a ripe plum."

There are two distinct methods of absorption; in one the blood spreads from the center to the periphery in an ever widening circle of dark granular blood. The sclerosed choroidal vessels are visible. Finally, the blood disappears and is followed by an abnormal pigmentation, often with a gray-white, closely adherent membrane, similar in appearance to a retinitis proliferans sheet. Central vision is greatly disturbed, often abolished. Widespread changes in the fundus may be coexistent, but sometimes they are absent.

In the other form, the hemorrhage is massive, involving not only the macular area but also a large contiguous region. In this type the retinal haze, which covers a larger area, passes progressively from the diffuse milky film to gray striations, fine yellow dots, gray stippling and sieve-like patches of whitish exudate. The blood absorbs throughout the mass, but most rapidly at the periphery where islands are formed, as the uneven absorption leaves thicker patches separated from the larger clot. The obscuring translucency of the retina is common. Retrogressive changes are varied. In some of these spontaneous hemorrhages, the vascular accident may have been anticipated because of the condition of the retinal and choroidal vessels, but too often one does not see the patient in advance, nor does one appreciate the danger signs. Quite obviously, the patient may be examined in any stage of the disease from onset to complete scarring, and for that reason I present this communication to the section.

A hemorrhage may form and disappear to be followed by others near the same place or separated from the original site before the retinal change becomes manifest, or retinal edema may precede the choroidal bleeding.

CASE 9.—Mrs. A. T., aged 65, came in complaining that for two weeks she had been conscious of an increasing defect in the vision of the left eye. The right eye was normal, except for a few opacities in the adult nuclear layer, and a compound myopic astigmatism.

Vision in the left eye was 3/200. There were many spicules of opacity in the adult nuclear layer (fig. 4, *1*). The disk was clearly and distinctly outlined with a broad central excavation, and the retinal vessels were normal. A large choroidal hemorrhage occupied an oval region, including the macula near its lower nasal side. There were a few isolated blood islands on a slightly milky film surface. Distributed over the hemorrhage were many fine, yellow-white dots.

Forty-eight days later the hemorrhage had steadily absorbed until now a broad arc surrounded a pale oval area from which the blood had disappeared (fig. 4, *2*). This seemed larger than it really was because the gray retina shaded some of the extravasation and also veiled the oval cyst. The macula was overshadowed by a black retinal flake.

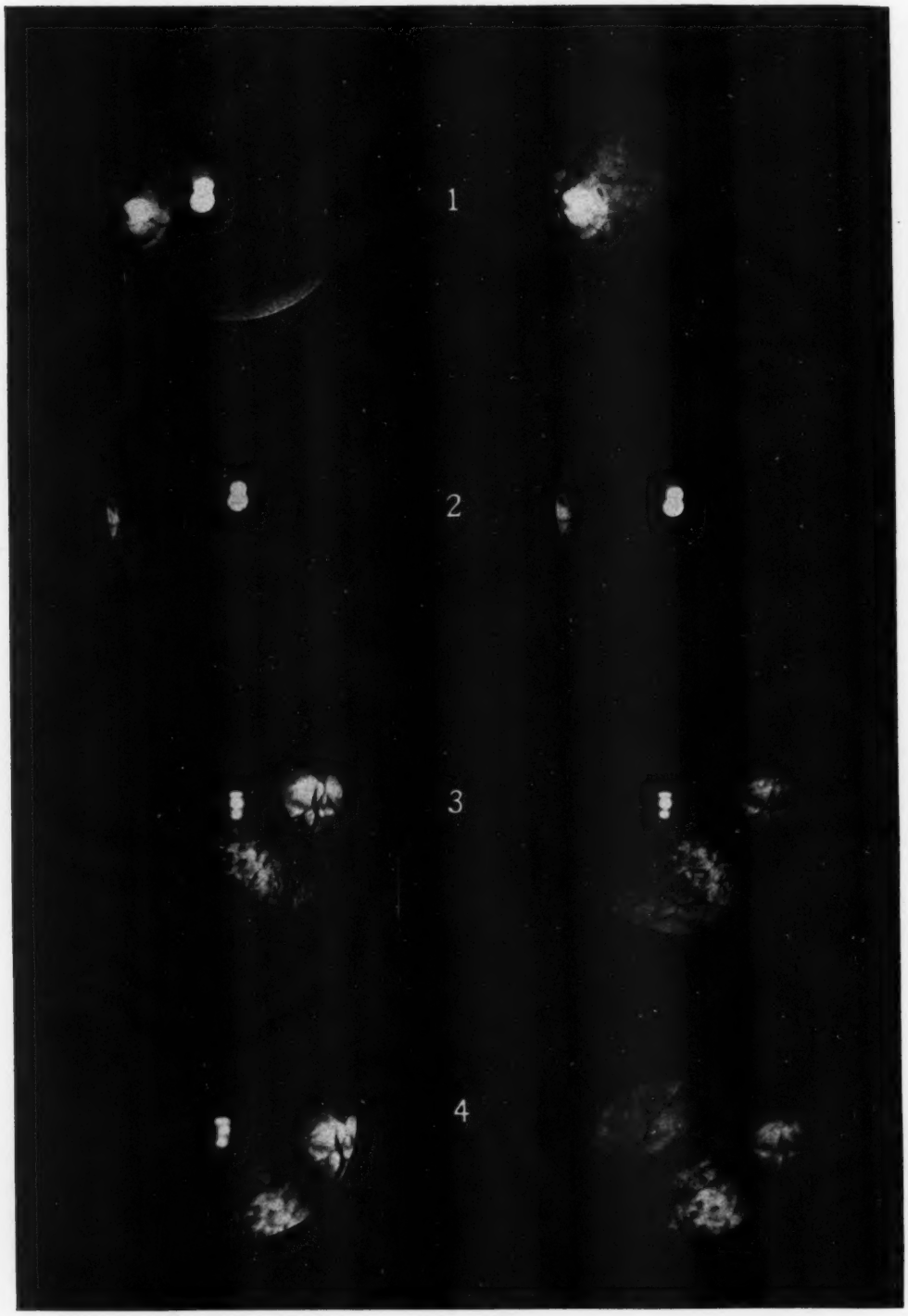

Fig. 4.—Spontaneous macular hemorrhage (*1* and *2*) and choroidal hemorrhage in a case of myopia (*3* and *4*).

CASE 10.—Miss H. S., aged 57, always highly myopic, noticed a blur before her right eye. This was ascribed by her attending physician to a detachment of the retina, and after five weeks in bed I saw her to find the condition as shown in figure 4, *3*. A delicate gray haze obscured the retina in the region of the macula, reducing the vision. The disk was oval with an inferior conus. Many patches of marked sclerosis of the choroidal vessel were scattered about the disk. A large one was seen below the macula.

Exactly three months later, it was noted that without any change in the sclerosis, a large irregular pyramidal area was outlined in dark red, choroidal blood (fig. 4, *4*). The oval center was cloudy gray, elevated and so translucent that the choroid was seen through its upper part. The angulations of a retinal artery over the cyst afforded an excellent guide to the degree of swelling and were especially worthy of careful observation.

CASE 11.—Mrs. A. B., aged 58, had worn glasses forty-six years when she suddenly discovered she was unable to read. For three days there had been a more marked decrease in sight.

Vision in the right eye was 1/200. Through thick, rather dense vitreous shreds the pale fundus was seen (fig. 5, *4*). The distorted oval disk was separated from a temporal staphyloma by a pigmented arc. No large choroidal vessels were visible. In the macular region was a large, red granular zone, over which a retinal vessel could be traced. Close scrutiny revealed a delicate, smoky gray haze toward the disk side of the hemorrhage. This varied considerably in depth and density. An oval below the center was the darkest and the thickest part of the hemorrhage.

Vision in the left eye was 1/200. The fundus was pale (fig. 5, *3*). The disk was drawn to the nasal side, showing a small oval and a wide temporal staphyloma. Several atrophic choroid spots formed a partial white frame to a bright red choroidal hemorrhage in the macular region. The outer edge of the clot was densest. The retinal vessels passed over it.

CASE 12.—Mrs. S. S., when 49 years of age, was seen with hyperpiesia, at which time her systolic pressure was 245. When she was 60 years old, she complained of a definite sensation of blurred vision in her right eye, which she stated had been present for at least three months.

Vision was 5/200 (fig. 5, *1*). The macula was distinguished as a dark, almost round area forming the upper part of the superior edge of a prominent, bulging, dark red hemorrhage which came from the choroid. The choroidal portion, which was seen through the thick clot, was circular in outline and granular in appearance. Over it, the retinal blood vessels passed. On the sloping sides of the protruding retina and clot were many glistening, yellow-white flecks of retinal exudate and a few streaks of superficial retinal blood. Where the hemorrhages streamed through the retina, the delimiting edge was gray, translucent, edematous retina. The surface of the most prominent portion of the clot was smooth.

About three months later, the macular circle was decidedly less distinct (fig. 5, *2*). A slightly oval, dark red spot about one-third disk diameter in size lay below and to the outer side of the macula and was the point of greatest projection as seen in the previous picture. The retina surrounding this point was much clearer, and small granular dashes of blood were seen in the adjacent choroid. The retinal exudate was less definite, and the retinal hemorrhages had practically disappeared.

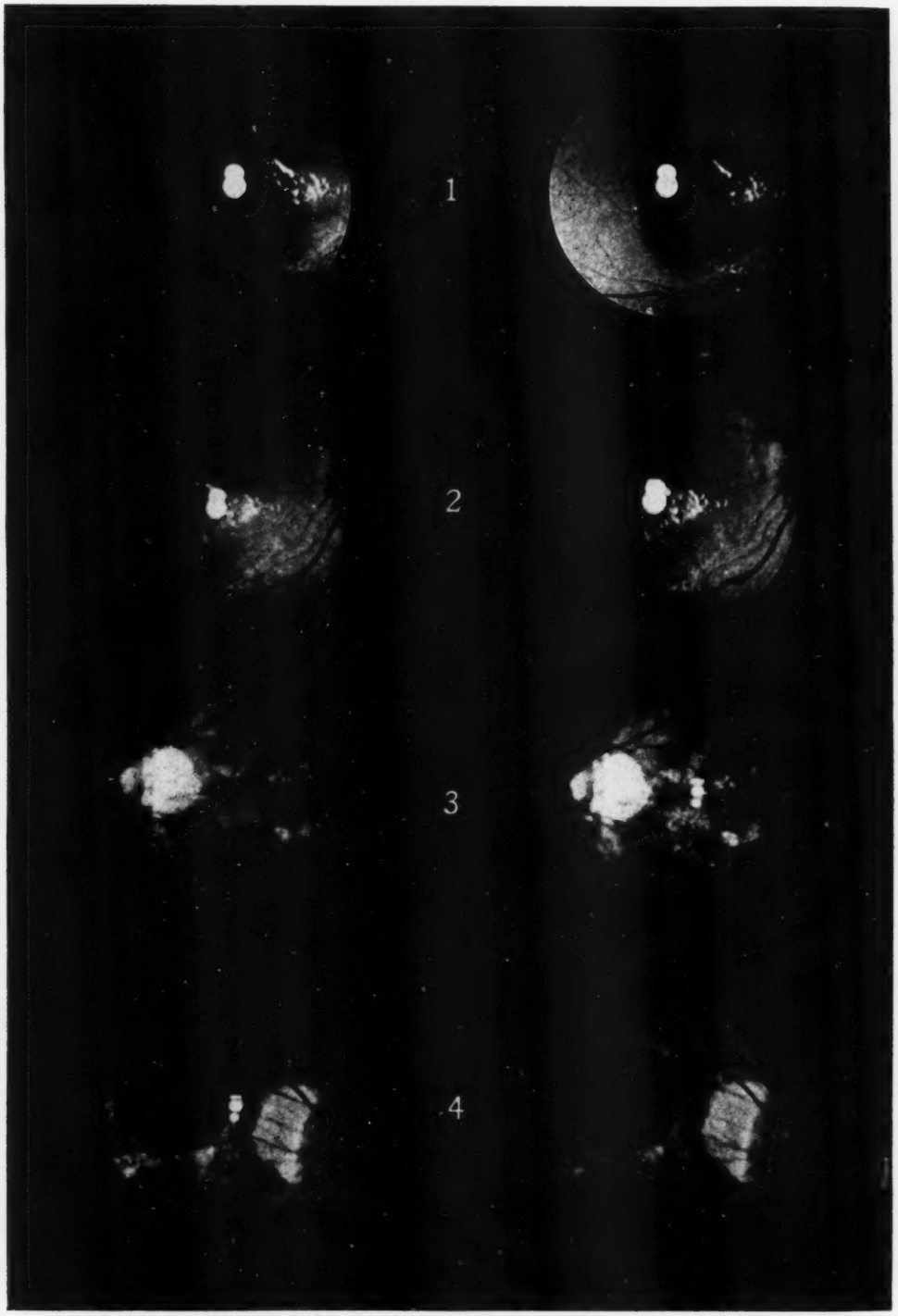

Fig. 5.—Choroidal hemorrhage in a case of myopia (*3* and *4*) and choroidal and retinal hemorrhage with hypertension (*1* and *2*).

DIFFICULTIES IN DIAGNOSIS OF CHOROIDAL HEMORRHAGE

The choroidal hemorrhage of the myopic patient is a curious disease, for great elongation of the posterior pole, extensive absorption of the retina and choroid and extreme sclerosis of all the choroidal channels without extravasation are seen; while in moderate stretching of the choroid, one may see the typical macular hemorrhage. Why this is so is difficult to explain, but it may be that in some cases the distention is so gradual that reparative processes prevent a tear in the vessels even when the retina and choroid are both destroyed in parts. In certain cases in which the large venous channels follow an unusual course, they may become wider near the macula and give the appearance of blood, while in others the angulation of the merging vessels may reflect the light so that the illuminated area looks like a hemorrhage. This is sometimes difficult to diagnose and is especially annoying because extravasations do occur under precisely the same conditions. This confusion is not, however, confined to myopic eyes, for I have seen cases of macular degeneration with marked choroidal sclerosis without hemorrhage or evidence of previous extravasation in hyperopia.

CASE 13.—A. C., a man, aged 25, who had worn glasses for eight years, noticed that for a month he had blurred vision.

Vision of the right eye was 1/200 $-14.00 = -2.00 \times 120°$ 20/70. The oval disk lay in the hollow of an annular staphyloma with normal retinal vessels entering into the central excavation (fig. 6, *1*). The choroidal and retinal pigment was unevenly distributed, giving a mottled background. A broad vena vorticosae formed a sharp angle near the lower temporal edge of the disk. The portion extending almost horizontally was especially wide as it curved beneath the macula. The channel seemed narrowed at the bend, but this was only the result of the angle at which it was photographed.

This is a condition often mistaken for an extravasation.

An especially annoying form of choroidal hemorrhage is seen every now and then after the successful removal of a cataract.

CASE 14.—Mrs. G. M., when 58 years of age, was operated on by me for removal of a cataract from the left eye, with uncomplicated healing and restoration of complete function. Nine years later she reported that there had been a gradual obscuration of objects for three weeks (fig. 6, *2*).

The disk was almost round with a broad inferior and temporal staphyloma. The retinal vessels were normal, and the choroidal circulation indefinite. A dark red hemorrhage covered the macular region. It seemed of uneven depth because the gray retina was only partly translucent and the portions beneath the clear retina seemed redder.

CASE 15.—Miss H. S., aged 78, had vision in her right eye of 2/200, which was improved to 20/70 with a $-14.00 = -3.00 \times 90°$. She showed a choroidal hemorrhage which was distinctive and yet most difficult to diagnose.

In the right eye (fig. 6, *3*) a small oval disk was found to the nasal side of a large, deep staphyloma. The broad collecting venae vorticosae dominated the picture. The horizontal vessel was shown with several smaller branches emptying

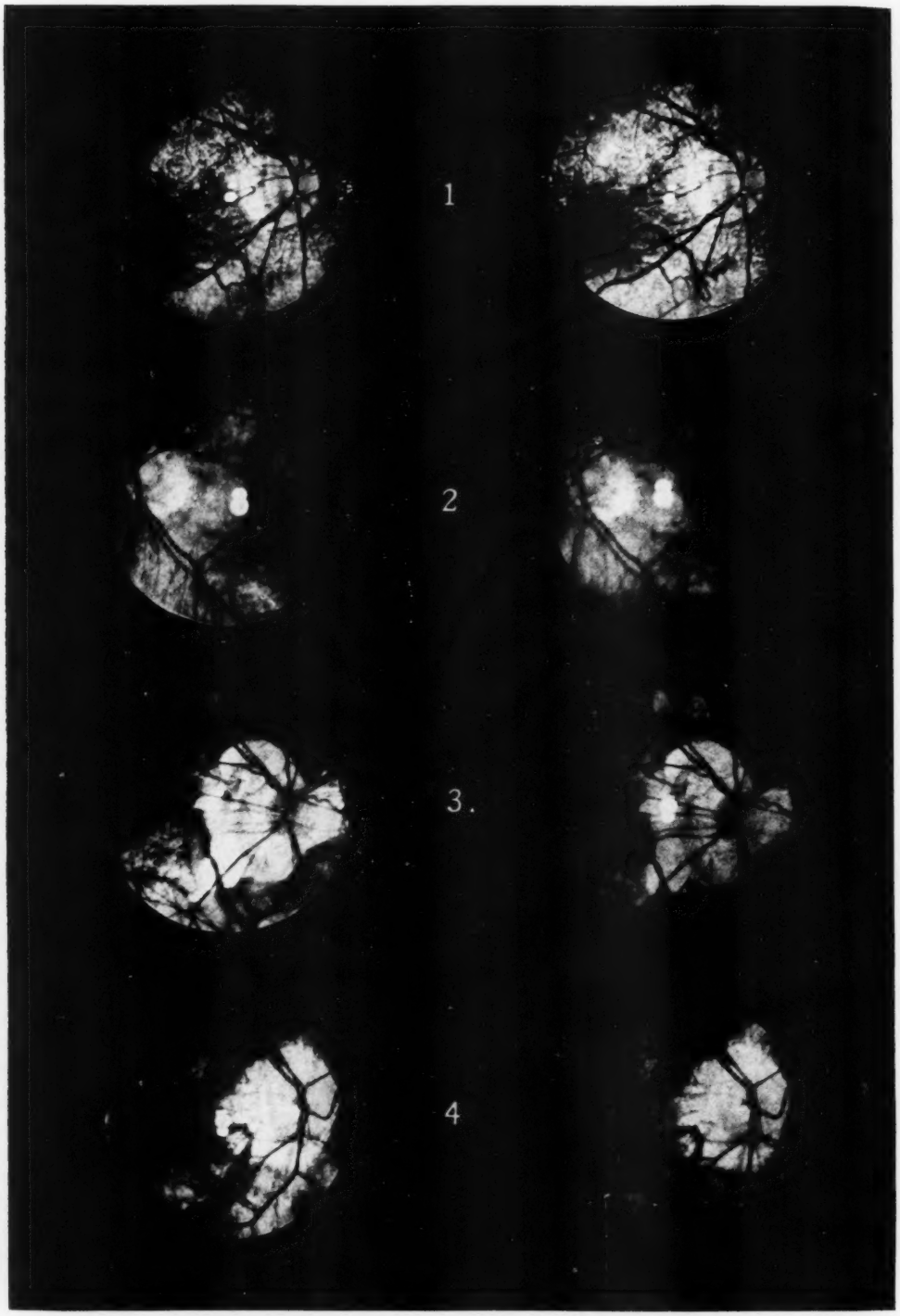

Fig. 6.—Choroidal pigmentation in a case of myopia (*1*); choroidal hemorrhage in a case of aphakia (*2*); choroidal hemorrhage in a case of myopia (*3*), and prominent venae vorticosae in a case of myopia (*4*).

into it from above. The widest channel came from the lower fundus; one broad branch was over the inferior edge of the staphyloma, the other was more to the temporal side. In the angle formed by their junction, almost directly opposite the disk was a round, red, granular choroidal hemorrhage over which a small retinal artery passed.

CASE 16.—Mrs. F. H., aged 35, had vision in the right eye of 6/200, improved with $-8.00 = -2.00 \times 75°$ to 20/20. From a temporal staphyloma, a cilioretinal artery emerged (fig. 6, *4*). The disk was oval and the retinal vessels normal. The choroidal circulation was so clear that the different layers of vessels were demonstrable. By the peculiar arrangement of two medium-sized choroidal channels and a local increase in the retinal pigment, the macular region was dark red. There was no hemorrhage.

Among the predisposing or exciting causes of choroidal extravasation is choroiditis, for few acute inflammations are unaccompanied by bleeding.

CASE 17.—Mr. T. R., aged 74, came in because of dizziness when he stooped. Blood pressure was 210 systolic and 110 diastolic; his radials and temporals were markedly atheromatous. He had albuminuria and myocardial degeneration.

Vision of the right eye was 16/200 (fig. 7, *1*). There was a rather widespread retinal edema, most easily seen in the photograph, about the nasal half of the disk. The arteries were stiffened and dented the veins where they crossed them. There were several flecks and dots of yellowish-white exudate, including some specks radially placed about the macula. A few superficial snow-bank opacities were fringed with retinal blood. There were striate and rounded retinal hemorrhages and deep, round, choroidal extravasations. The choroidal channels were visible in the nasal portion of the field.

CASE 18.—Mr. McK. was first seen Dec. 17, 1928, when he was 38 years old. Vision of the right eye was 20/70 (fig. 7, *2*). The disk was clearly and distinctly outlined, and the retinal vessels normal. Surrounding the macula was an elevated, gray circle partially fogging the dark center. There were several areas of choroidal atrophy, some almost as long as the disk and others mere yellowish specks.

The choroidal degeneration had steadily progressed so that there was distinct atrophy, and the migration of pigment was more evident (fig. 7, *3*). The retinal haze had an upward point which was outlined by dark choroidal hemorrhages. The clot seemed wrinkled, but only because the translucent folds in the overlying retina decreased visibility.

Pathologists have found choroidal hemorrhages in papilledema, but in life the other features of the fundus are so absorbing in interest that one may fail to recognize a small isolated extravasation. One such case is shown to illustrate its appearance.

CASE 19.—A man, aged 36, had a tumor of the temporal lobe. Vision of the left eye was 20/40. There was papilledema with vascularized exudate cap, full veins, obscured arteries and concentric folds in the retina, as shown in figure 7, *4*. Below the macula was a dark red, round hemorrhage in the choroid. The overlying folds in the retina were accentuated by shadows and appeared like dark streaks. The retinal vessels passed over the blood.

Fig. 7.—Choroidal hemorrhage in a case of arteriosclerotic retinitis (*1*), in a case of choroiditis (*2* and *3*) and in a case of brain tumer (*4*).

Choroidal hemorrhage must be differentiated from subchoroidal, subretinal, retinal and preretinal blood; venae vorticosae and large choroidal vessels; choroidal neoplasm; pigment after prolonged hypertension; the pink reflex found in myopic eyes without hemorrhage; traumatic pigmentation of the fundus and certain pigment collections after choroiditis.

Preretinal blood covers the retinal vessels, whereas retinal blood is striate and the superficial vessels rarely, if ever, course over it. This contrasts with choroidal hemorrhage, which is most often globular, and even when striate the granular character predominates; it is deep and the retinal blood vessels pass over it. A prechoroidal, or subretinal, extravasation is determined by stereoscopic examination or photographs showing its position. This is found as small, round spots such as are seen following thrombosis or nephritis.

CASE 20.—Mrs. B. J., aged 59, had complete ptosis with marked divergence of the left eye. Vision was 1/200 (fig. 8, *4*). The round disk was completely encircled by a zone of choroidal atrophy of unequal width with an unevenly pigmented edge. The vessels passed over the sclera to dip abruptly into a large glaucoma cup. All of the retinal vessels were remarkably straight. Through the thin retina the choroidal channels were easily traced. The dark region above the macula was not a hemorrhage, but was caused by the retention of the retina and an especially thick group of choroidal channels.

CASE 21.—Mr. J. H., aged 75, had vision in the left eye of 2/200 (fig. 8, *3*). The disk was vertically placed in a ring of choroidal atrophy. The retinal vessels were not noteworthy, while the choroidal ones were readily seen. An irregular, ovoid, white patch of choroidal and retinal loss almost twice the size of the disk occupied the macular region. A retinal vessel passed along its upper margin. Scattered spots of pigment were in the retina just beyond the lower edge. A large, diamond-shaped clump of pigment and many fine granules were spread over the atrophic area.

CASE 22.—Mrs. M. R. was 57 years of age when the picture shown in figure 8, *2* was made of her right eye. Vision was 2/200 unimproved with a $-13.00 = -3.00 \times 165°$.

In a wide nasal staphyloma the oval disk was easily separated by its color from the choroid atrophy. Two dark patches one above and the other below the disk were deep red; the upper was retained retina, while the lower one was a knuckle of a choroidal vessel extending over the staphyloma. The dark masses over the white macular area were pigmented accumulations similar to those about the upper margin of the destruction but so thick and so superficial that they lay on top of the scar.

CASE 23.—A woman, aged 46, who had never worn glasses complained of poor sight. Vision in the right eye was 4/200, unimproved with a $-12.00 = -3.00 \times 30°$ (fig. 8, *1*).

A wide staphyloma surrounded the disk. A few choroid vessels were seen on the floor, but most of the choroid had disappeared in the stretched zone. The disk was small and the retinal vessels normal in their straight course. The lower nasal edge of the staphyloma merged into a small patch of old choroiditis with heaped-up pigment. The great choroidal channels were not visible. An oblique tear in the

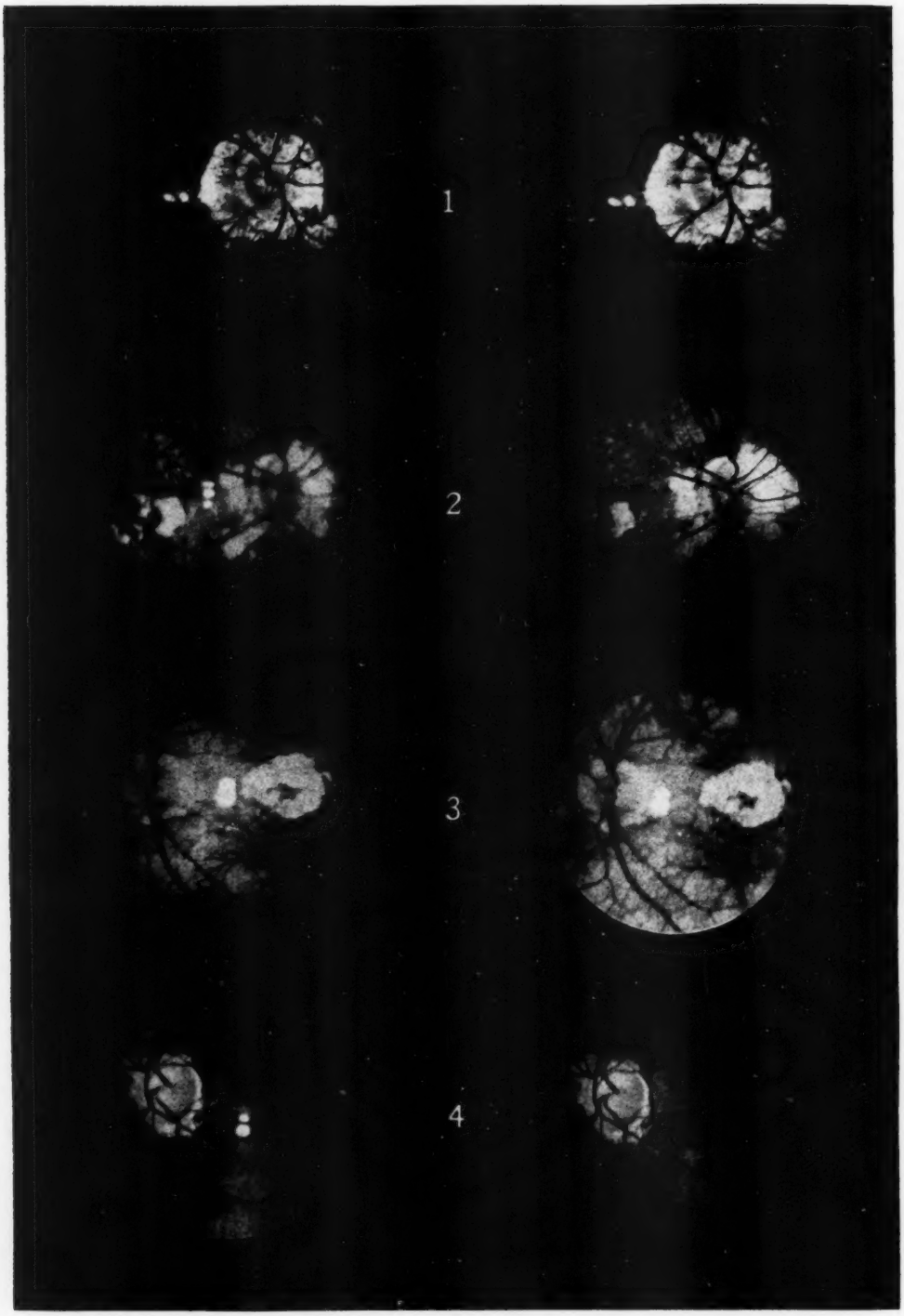

Fig. 8.—Glaucoma and myopia without hemorrhage (*4*); retinochoroiditis (*3*); choroidal pigmentation in a case of myopia (*2*), and myopia with macular tears (*1*).

choroid extended from the temporal side of the staphyloma down and out. In places the rent was complete, showing the white sclera; in others, it was partial, causing a decrease in the intensity of fundus pink. A middle-sized choroidal vessel was seen between two white spots.

CASE 24.—Mrs. C. D., aged 72, had a bilateral lesion. Vision in the left eye was 20/200 (fig. 9, *1*). The small disk was outlined by a nasal conus and a temporal pigment line. The reflex was pink and yellow where the retina was thinned. An area 3 disk diameters in size included the macula within its confines. The floor of this region was uneven, but in several places the choroidal channels were seen. There was an almost round, dark reddish-black plaque lying near the center of the atrophied choroid. This was a pigmented mass.

CASE 25.—S. G., a man, aged 72, had noticed a steady decrease in vision for two years. His systolic pressure was 200.

Vision in the left eye was 3/200, with $+2.25 \times 165°$ 6/200. An extensive, white choroidal exudate occupied much of the posterior pole and encircled the disk except on its temporal side where it was adjacent to comparatively normal retina (fig. 9, *2*). In the center of this retinal area was an oval of destroyed choroid with splotches of pigment on its surface. The retinal vessels were negative and passed over the choroidal atrophy.

This exudate could be mistaken for a large choroidal hemorrhage.

CASE 26.—A woman, aged 52, had vision in the right eye of 20/50 (fig. 9, *3*). The entire fundus was pale, the choroid easily seen and the disk scarcely distinguished by color from the retina. In the center of a darkened ring an oval, granular-appearing, depressed, whitish macular scar was seen. There were no hemorrhages.

CASE 27.—E. P., a 62 year old woman, had a thrombosis of the central retinal vein of the left eye five years before consulting me. At the present examination a tangle of vessels projected over the disk (fig. 9, *4*). These were full with some irregularity of caliber. The change in lumen was more marked in the arteries where in some places there was real beading. All about the macula there were many minute aneurysms. The macular scar consisted of two pigmented rings; the blacker was the smaller and the more central, the outer was more line-like, although the edge was broken below.

When differentiating between choroidal pigment and choroidal blood, one should remember that the pigment is darker, more homogeneous and more irregular in outline with no great vacillations from time to time. Choroidal blood is always a shade of red from pink to almost chocolate black. It is granular when thin, and only homogeneous when thick. Usually it has a rounded outline, but it may be circinate or even striate.

The distinction between the dark spots found in hypertension and choroidal blood is made after due consideration of the points just mentioned, and the fact that the round chocolate colored spots of hypertension neither change in size nor migrate and are always accompanied by arteriosclerotic vessels, while in choroidal hemorrhage it is not unusual to find that the blood disappears from one part and a new extravasation appears in another. This increase and decrease in size

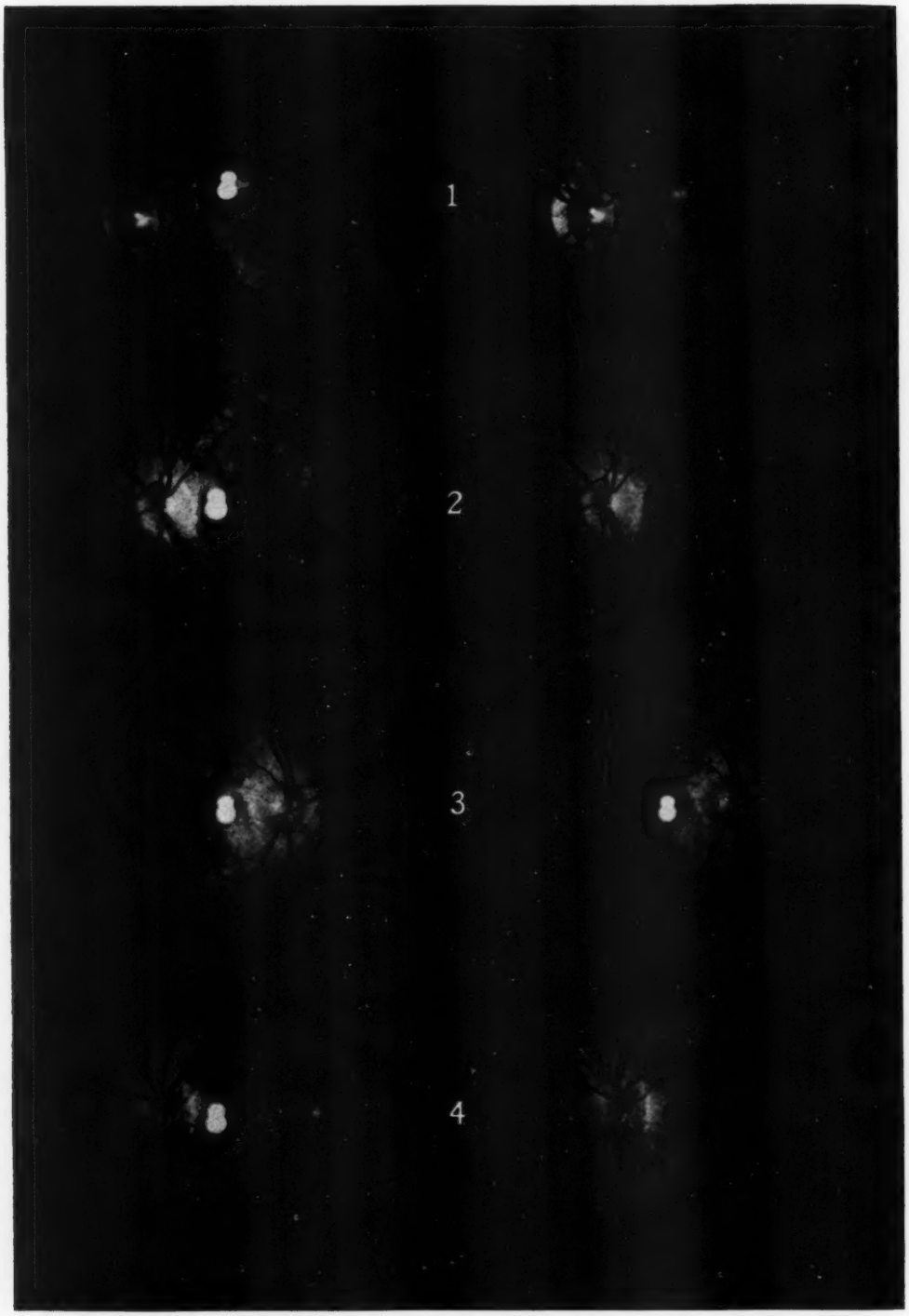

Fig. 9.—Central retinochoroiditis (*1*); choroidal exudate with macular degeneration (*2*); macular degeneration (*3*), and retinal and choroidal pigmentation after thrombosis (*4*).

of the individual hemorrhage may be extremely difficult to measure, for even in photographs one sometimes has to magnify the print before making a definite decision. Certainly the ordinary ophthalmoscopic examination is not sufficient in all cases.

CASE 28.—Some of these points are revealed in the photographs of Miss H. M., who first came under observation when she was 41 years old, twenty-one years before the present examination. At that time the vision with a low degree of compound myopic astigmatism correction was 20/20 in each eye.

On April 24, 1930, when she was examined, she stated that for three years she had had diabetes. The vision in the left eye was 20/200 (fig. 10, *1*). The hemorrhages, dark, flat extravasations about the macula, were beneath the retinal vessels. There were marked perivascular changes, especially over the pale disk. A vitreous opacity gave a smoky effect, noted as a cloud in the photograph.

On May 28, 1930, vision was 20/70 (fig. 10, *2*). The vitreous cloud was greatly lessened and, although some choroidal blood had disappeared, there was a large V-shaped clot on the margin of a partly depigmented choroidal area below and about the macula.

On Sept. 5, 1930, vision was 20/200 (fig. 10, *3*). Two areas attracted attention; one was the gray elevated macula with dark blood ring and the other a mass of whitish-yellow retinal exudate.

On Oct. 8, 1930, the transitory character of the exudate, the disappearance of old hemorrhages and the formation of new ones in a different location were seen (fig. 10, *4*). The retinal vessels were traced over the remaining clot to the degenerated macula.

Choroidal blood is smoother, often darker in color and transilluminates better than a choroidal tumor. Migration of choroidal pigment following an injury might to an untrained observer be misinterpreted as choroidal blood, but the color and characteristic structure associated with the general changes in the fundus are sufficient to distinguish one from the other. Sometimes the heaped-up pigment of choroiditis and chorioretinitis simulates in appearance as well as distribution choroidal blood, but a careful analysis of all factors is sufficient to establish the correct diagnosis.

CONCLUSIONS

The significance of choroidal hemorrhage may be divided into several groups of clinical conditions: what it means after an injury, when it is spontaneous, when concomitant with choroidal inflammation, when present in myopic elongation of the eyeball, as a causative factor in the destruction of eyes following intra-ocular operations, when an integral part of arteriosclerosis, of hypertension, of diabetes and the various blood dyscrasias, as well as when it makes its rare appearance in the papilledema of brain tumor.

After a trauma, it means a rupture of the choroid, which may or may not involve the macula.

When spontaneous, it is a grave symptom whether in arteriosclerosis. hypertension, diabetes, blood dyscrasias like pernicious anemia and

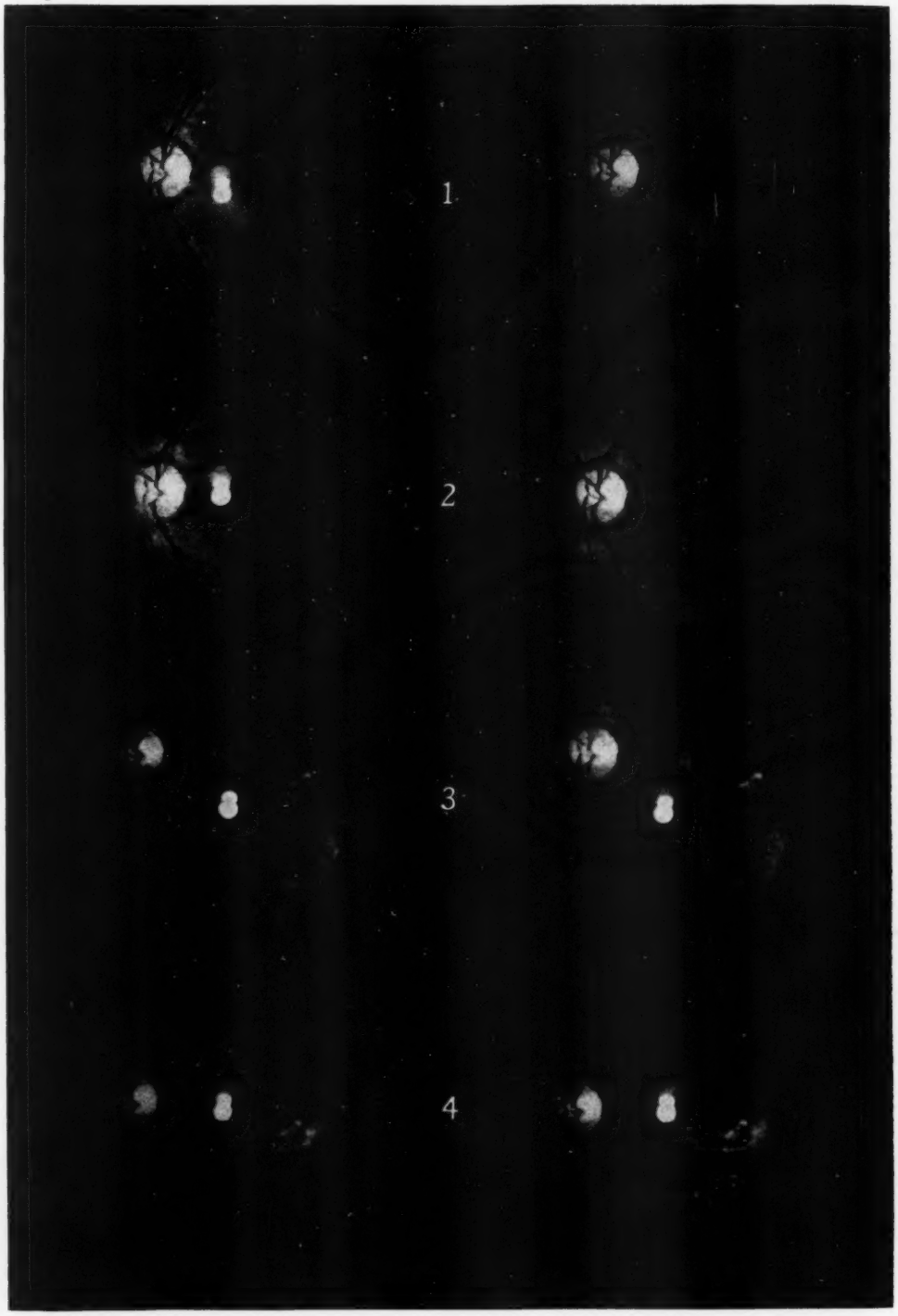

Fig. 10.—Choroidal hemorrhage in a case of macular degeneration, arteriosclerosis and diabetes.

leukemia or without demonstrable cause. The more centrally the lesion is placed, the more destructive of function. Even a small choroidal hemorrhage may be the forerunner of an expanding ring of blood, and I have seen it as the first sign of a rapidly advancing vascular death.

In practically all types and cases of choroiditis, some choroidal blood is present during the course of the disease. Old hemorrhages disappear, and new ones develop near the border of the spreading lesion.

Hemorrhage in the macular choroid of myopic persons always reduces vision, and is evidence of choroidal stretching.

When the eye bleeds profusely, after an intra-ocular operation, the probability that it comes from the choroid is sufficient to give one great concern.

When discovered in the routine examination of the fundus in brain tumor, choroidal hemorrhage seems to be devoid of significance, although even here when it involves the macular region destruction assumes an important rôle.

And finally, when choroidal hemorrhage breaks through the retina and into the vitreous, it is an indication of a large extravasation, which in my experience is either of traumatic or circulatory origin.

ABSTRACT OF DISCUSSION

Dr. Everett L. Goar, Houston, Texas: As Dr. Burch was unable to attend this meeting I have been asked to substitute for him in opening the discussion of Dr. Bedell's paper, so the following discussion will embody some of Dr. Burch's ideas on the subject, together with my own.

Dr. Bedell, in his characteristically thorough manner, has so completely covered his subject that he leaves little room for additions and none for subtractions. He has not only discussed the subject from every possible angle, but has shown photographs illustrating the various stages of choroidal hemorrhage, from the earliest extravasation of blood to the final stage of scarring, with the destructive effect on the overlying retina.

One of the most interesting facts that the author has shown is that the so-called "senile degeneration of the macula" is in truth a choroidal hemorrhage seen in the late stage when the blood is no longer visible, as such, but has left its mark plainly to be seen, if properly interpreted.

Hemorrhages from the choroid or in the macular region, even when small, are more destructive of vision than retinal hemorrhages, and they are more slowly absorbed. The fact that the outer layers of the retina are nourished by the choroid, and that the layer of rods and cones lies adjacent to the lamina vitrea perhaps accounts for the greater visual destruction. As spontaneous choroidal hemorrhage usually occurs in persons who have a marked sclerosis of the choroidal vessels, absorption of extravasations is necessarily slow.

Dr. Burch suggests that in operating on an eye with increased tension, either for glaucoma or cataract, measures that will reduce the ocular tension as well as the muscular hypertension should be used. He sometimes resorts to venesection before operation in patients with hypertension. He believes that making the section deliberately, allowing the aqueous to escape slowly and avoiding undue trauma with the fixation forceps, are important in avoiding this most distressing complication.

Dr. Burch has sent three slides, with his description of them, showing photographs from two cases of choroidal hemorrhage. The first is that of a hypermetropic patient, aged 51, who suddenly lost the vision in the right eye. Ophthalmoscopic examination showed an extensive hemorrhage in the macular region which was definitely choroidal in origin. There were no other fundus changes. There is no history of trauma, the blood pressure was not abnormally elevated, and physical examination gave otherwise negative results. The Wassermann reaction of the blood was positive. After ten months of systemic treatment the macula showed an oval discrete atrophic patch with a fairly sharp border like a macular hole. No new lesions have developed in either eye. Why and how then could this single hemorrhage, obviously of choroidal origin, be explained unless on the basis of a forgotten trauma? It is not easily explained as an acute syphilitic lesion since during twenty months of observation it was the sole tertiary manifestation of the disease.

The last picture is one of senile macular degeneration in which there was an extensive hemorrhage. This resulted in retinal detachment of the entire macular area with a retinal tear followed by gradual reattachment. The patient, aged 79, showed senile macular degeneration in each eye. The slide is made from a photograph of the fundus which was taken about one year after the vision first became involved. The tear is still present but the reattached macular elevation argues for an adhesive process following hemorrhage with degeneration, rather than a true inflammatory process.

Dr. William H. Wilder, Chicago: The pictures exhibited by Dr. Bedell are clear enough to show the vascular changes that are the reason of the hemorrhages of the retina as well as those of the choroid.

It is true that most frequently, because of an unbroken pigment layer of the retina, one does not have an ocular demonstration of the sclerosis of the choroidal vessels like that seen in the retinal vessels, but the inference is justified that whenever marked retinal arteriosclerosis is present sclerosis of the choroidal vessels probably exists as well. This would account for hemorrhages of the choroid such as those shown in the illustrations.

I noticed that the camera has recorded faithfully some of the earliest signs of retinal arteriosclerosis such as diminished caliber of some of the arteries and tortuosity of some of the finer endings of small arteries such as terminals of vessels supplying the macular region. An early recognition of such vascular changes is valuable to the internist as well as to the opthalmologist, because they furnish presumptive evidence of arteriosclerotic changes in other parts of the body, particularly the central nervous system.

An accurate photographic record of the condition of the vessels such as shown in these illustrations would be useful for future reference in any case.

ACQUIRED CYST OF THE OPTIC DISK

JOSEPH LEVINE, M.D.
NEW YORK

The patient, A. L., is a 14 year old girl, a deaf-mute with retinitis pigmentosa. I have seen her at monthly intervals for the past three years. During the summer vacation in 1931, she was not seen until October, after an interval of four months. At this examination there was seen in the left eye for the first time a gray, tubular, cystic mass protruding from the optic disk at its nasal margin (fig. 1). Since then it has grown longer and thinner and projects farther into the vitreous, at present to a distance of 5 diopters. The base seems to be firmly attached at the disk, and the cyst itself moves from side to side like an inflated tubular balloon which is tied down at one end. There is a knoblike protrusion on the original cyst which can be called a daughter cyst, as it were. It is pearl gray, and the wall seems fairly thin; there are no blood vessels visible on the cyst. There are no retinal lesions in either eye except for the retinitis pigmentosa which is fairly well advanced. The media are clear, and there is no evidence of uveitis. The vision is 20/40 plus in each eye and is not improved by glasses. The fields show the typical contraction for form and colors expected in a case of retinitis pigmentosa, but they are not exact because of the obvious difficulty in obtaining accurate fields of vision from a deaf-mute.

The patient's personal and family history as stated in the records of the Institute for Improving the Condition of Deaf Mutes is as follows:

The parents were married nineteen years ago. They have three children: Morris, 18; Anna, 14 (the patient under discussion) and Aaron, 6. The two brothers are reported to be very bright. The older one works during the day and attends college at night. All members of the family are reported to be in good health.

Anna was born after a normal pregnancy, at the Greenpoint Hospital in Brooklyn; no instruments were used. Her mother stated that from the beginning she "had a cold in her eyes" and had sniffed a good deal, and that for some time boric acid had been sprayed into her nose. When the child was about 3 months old, she was thrown from a wagon in which she was riding with her mother. While she did not appear to be hurt at first, convulsions developed the following day and continued to occur for some time afterward. The child cried a great deal, and abscesses also developed in the ears. At the age of 3 years she fell into a cellar and injured her ear. She was treated at the Post-Graduate Hospital, where her mother also took her for treatments in the Mental Hygiene Clinic before the child entered the school at the Institute.

The mother comes from a family of six children, all of whom are reported to be in good health and of normal mentality. The maternal great grandmother became blind in later life; the cause was unknown. The father has nine brothers and sisters, all of whom, including their families, are reported to be well. Mrs. L. stated definitely that there is no epilepsy, mental deficiency or idiocy in either of

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Read before the Section of Ophthalmology, New York Academy of Medicine, March 21, 1932.

the two families, nor does she know of any one suffering from tumors. She also stated that she and her husband are not related.

Dr. Isador Abrahamson examined the patient, and his report is as follows: "Neurological examination gave practically negative results aside from the ocular findings and the many pigmented spots over the body. In the absence of any neurologic abnormality, even though the ocular findings are rather suggestive, I should not care to make a diagnosis of tuberous sclerosis at present."

Dr. Joel Schweig examined the skin of the patient and found no evidence of adenoma sebaceum or of Recklinghausen's disease, although there were many pigmented moles on the body.

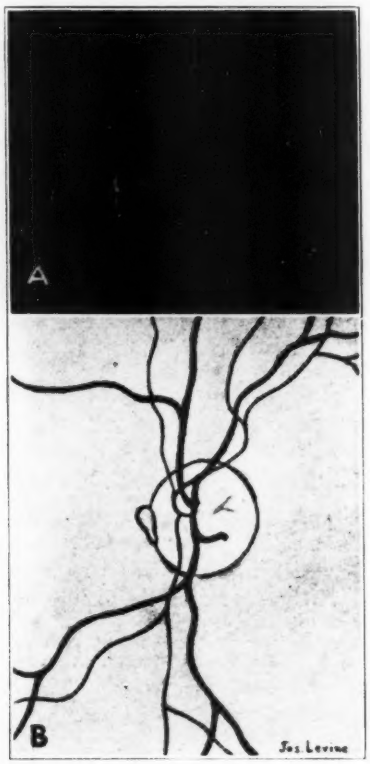

Fig. 1.—*A*, photograph of the fundus showing a cyst of the optic disk; *B*, drawing of the fundus showing the cyst.

In differentiating between congenital and acquired membranes and cysts, Durst [1] made the following points:

1. Congenital cases usually show other congenital anomalies.

2. Acquired cases often have evidences of recent or old inflammatory processes in the deeper structures of the eye.

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1. Durst, Alfred: Ueber Geschwülste und geschwulstähnliche Gebilde der Papilla nervi optici, Inaug. Diss., Leipzig, Robert Noske, 1909.

3. If blood vessels are present in these membranes or cysts, one finds that in the congenital cases they are empty strands, but in the acquired cases they often contain blood.

4. In congenital cases there are often amblyopia and strabismus.

In the case under discussion, one observes that all the characteristics of an acquired cyst are present. There are no other congenital ano-

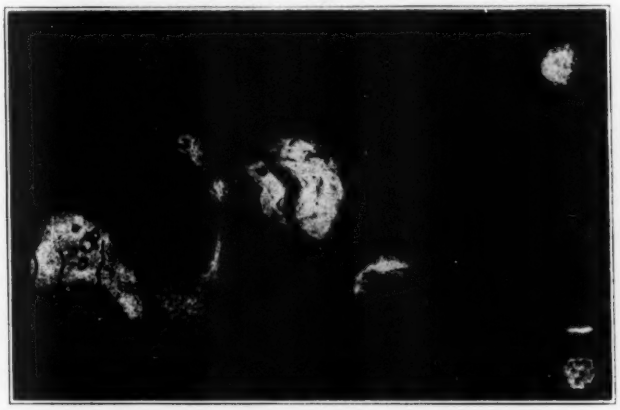

Fig. 2.—Raised tumor masses of the optic disk proper in a case of tuberous sclerosis. (From van der Hoeve: Arch. f. Ophth. **105**:880, 1921.)

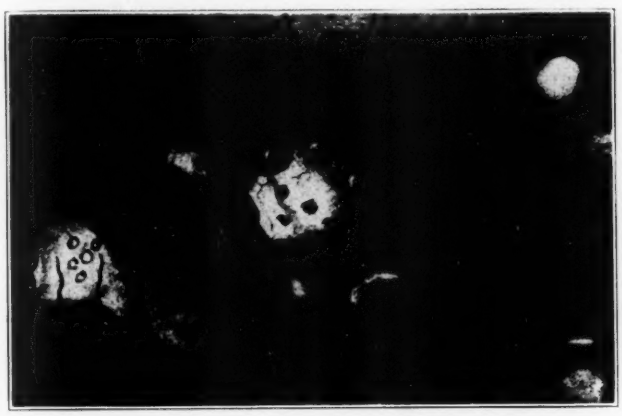

Fig. 3.—Same as figure 2.

malies, there is no amblyopia or strabismus, and there are evidences of intra-ocular changes (retinitis pigmentosa). Accordingly, in determining its pathogenesis, one has two conditions to consider, (1) tuberous sclerosis and (2) retinitis pigmentosa.

Tuberous sclerosis, also called gliosis, consists in a development of nodules and larger nodes on the surface of the brain due to a local

proliferation of glial tissue. It usually starts early in life, before the age of 25, but cases have been reported in which the manifestations became evident later. It is most common in families with a history of familial degeneration, other tumor growths or syphilis. The child may be very bright at school but may gradually find it more and more difficult to keep up with his class work; then dizzy spells may develop, followed by epileptiform attacks, idiocy and, finally, death.

The tumor masses in the brain cortex consist of glial tissue (connective tissue or supporting tissue of the nerve fibers). However, there are similar proliferative masses in other parts of the body, e. g., the

Fig. 4.—Flat tumor masses of the retina in a case of tuberous sclerosis. (From van der Hoeve: Arch. f. Ophth. **105**:880, 1921.)

skin, the peripheral nerves, the heart, the thyroid gland and the breasts. In the skin they manifest themselves as adenoma sebaceum, also called nevus sebaceus. In the cutaneous nerve fibers they are neurofibromatosis or Recklinghausen's disease. Moreover, these proliferative masses occur in the eye, and here they are seen in two main forms: (1) as raised tumor masses of the optic disk proper (figs. 2 and 3), and (2) as flat tumors of the retina (figs. 4 and 5).

Van der Hoeve [2] had the good fortune to examine these cysts of the nerve and flat masses of the retina microscopically. He found that the

2. van der Hoeve: Ueber Tuberosclerosis, Arch. f. Ophth. **105**:880, 1921; **111**:1, 1923.

cysts protrude through the internal limiting membrane, and their walls consist of fibers and cells (fig. 6). These cells have much cytoplasm, large nuclei and nucleoli and some degenerative forms. They arise from the first retina anlage and are neurocytes that have not become differentiated into glia cells or ganglion cells. The entire swelling is edematous, part of it containing serum and part blood cells; in some places there is also blood pigment, and at times groups of white cells which are indicative of inflammation. There are few blood vessels. E. Fuchs agreed with van der Hoeve in believing that these masses arise from cells which, during the development of the retina, remained as undifferentiated "Baumaterial."

Fig. 5.—Same as figure 4.

In the retina these masses present themselves as very small outgrowths or just as a thickening of the nerve fiber layer which may vary up to large swollen masses. All these are made up of the same cells and fibers as the cystic masses at the papilla and all arise from the nerve fiber layer, or occasionally from the ganglion cell layer. In many places there are seen just thickenings of the nerve fiber layer consisting only of fibers and no cells. Furthermore, there are evidences of inflammation in the ciliary body where one sees small, lymphocytic infiltrations; there may be perivasculitis, vitreous membranes and newly formed blood vessels. The question of the presence of inflammation is interesting—is it the cause of the swelling or are these masses inherently prone to

inflammatory reactions? Every one of the masses has some inflammation even though in most of them it is slight, and van der Hoeve feels that the masses are the result of the inflammation.

From the tumor mass at the papilla small particles may drop off and float in the vitreous. These cystic masses may be very large; when too full they break and their fluid goes into the vitreous. If the tear in the cyst wall is small, it closes and the cyst refills only to break again. If the tear is too large, the cyst does not refill. Sometimes there is blood in the cyst, and its fluid level may be seen to vary from day to day.

Fig. 6.—Microscopic section of the cystic mass of the optic disk protruding through the internal limiting membrane. (From van der Hoeve: Arch. f. Ophth. **111**:1, 1923.)

Since Bourneville, in 1880, first made the diagnosis of "sclerose tubéreuse des circonvolutions cérébrales," tumor masses have been found not only in the brain but also in the skin, the nerves, the breast, thyroid gland and heart. In the latter they are subendocardial, congenital rhabdomyomas. In 50 per cent of the cases of congenital cardiac myomas, tuberous sclerosis is found to be present at autopsy. These patients die at a young age as a rule, often before the symptoms of tuberous sclerosis are apparent. In the other organs, the tumor masses are subcapsular and are leiomyomas, lipomas, hypernephromas, fibromas, angiomas, sarcomas and mixed tumors.

In the skin, the presence of adenoma sebaceum is fairly frequent in cases of tuberous sclerosis. The skin lesions are present on both sides

of the nasolabial folds, the brow and the chin and at times on the neck. They are discrete or confluent, pinhead-sized, slightly elevated, yellowish or reddish, translucent, waxy papules. They are of congenital origin but may not appear until adolescence and are often associated with other congenital anomalies such as spina bifida, ectopia testis, etc. There are two types of adenoma sebaceum, the Balzer type and the Pringle type. In the Balzer type the sebaceous glands are predominantly involved and are hyperplastic, while in the Pringle type the blood vessels are mainly involved and there is hypertrophy of the gland lobules with dilatation of the blood vessels; they are really nevi. According to Jacobi, the presence of adenoma sebaceum of the Pringle type, i. e.,

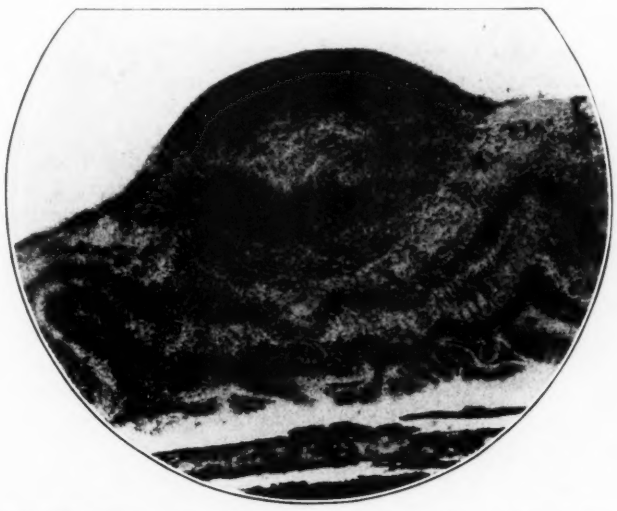

Fig. 7.—Microscopic section of the mass in the retina. (From van der Hoeve: Arch. f. Ophth. **111**:1, 1923.)

nevus sebaceous, would determine the differentiation of persons with ordinary epilepsy and idiocy from those with tuberous sclerosis.

As regards the presence of nevi along the course of the nerves, or neurofibromatosis (Recklinghausen's disease), it is considered by Bielschowsky, Orzechowsky and Nowicki that neurofibromatosis is the same as tuberous sclerosis except that in one the swellings are peripheral and in the other central. They speak of neurinomatosis centralis, peripheralis and universalis. Furthermore, in two cases of neurofibromatosis van der Hoeve saw lesions in the fundus similar to those seen in tuberous sclerosis.

In all the cases studied by van der Hoeve, one family is of such interest that the history is briefly reviewed here. The father was operated on for myosarcoma of the stomach. The mother had thirteen preg-

nancies, nine children being born alive. Of these nine, three had tuberous sclerosis and died; one child was in an insane asylum and had tuberous sclerosis; one had epileptiform attacks, and another was beginning to have these attacks. One child had no signs as yet, but in the fundus there was a mass below the disk typical of those seen in cases of tuberous sclerosis; the children with tuberous sclerosis had no lesions in the fundus. Van der Hoeve considers this of the utmost importance, because it emphasizes the fact that not in all patients with tuberous sclerosis are there ocular findings; however, in one patient these findings were the first manifestation of a later developing tuberous sclerosis.

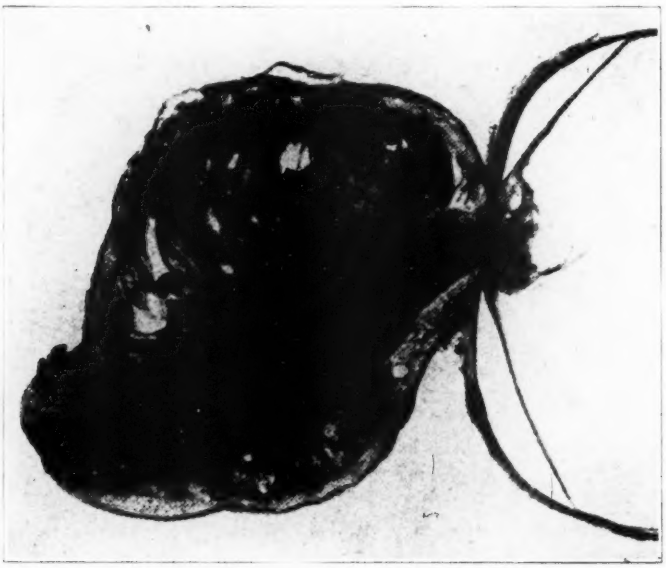

Fig. 8.—Cyst of the optic disk in a case of glioma of the optic nerve. (From Verhoeff, F. H.: Tr. Sect. Ophth., A. M. A., 1921, p. 146; Arch. Ophth. **51:** 120 [March] 1922.)

So much for the diagnosis in my patient of tuberous sclerosis, which cannot yet be made with any definite assurance of its accuracy. However, the patient has retinitis pigmentosa, of which diagnosis there can be no doubt. In retinitis pigmentosa there is degeneration of the neuroepithelium with a marked gliosis. Neuroglia cells are seen in the nerve fiber and ganglion cell layers, and there is a definite neuroglia layer in front of the optic disk which is responsible for the typical waxy appearance of the disk as seen with the ophthalmoscope. Furthermore, on pathologic examination, Verhoeff,[3] in one of his cases of retinitis

3. Verhoeff, F. H.: Microscopic Observations in a Case of Retinitis Pigmentosa, Arch. Ophth. **5**:392 (March) 1931.

pigmentosa, saw small accumulations of neuroglia cells immediately beneath the hyaloid membrane and also small cysts in the outer layer of the retina near the internal limiting membrane. These were lined by a membrane and contained serum. "Various stages in the formation of these cysts could be observed, and it was seen that they were due to infolding of the external limiting membrane and altered neuroepithelium resulting from contraction of the newly formed neuroglia" (Verhoeff,[3] fig. 8).

Verhoeff, in a personal communication, stated:

> I have never seen in a case of retinitis pigmentosa such a cyst as you describe. If it arose at the margin of the disk, it possibly could be similar to the cysts I found in the retina, but if it arose from the tissue of the disk, this could not be so. If it arose in the tissue of the disk, I should assume that it was due to gliosis associated with marked cystoid change. Hyperplastic neuroglia everywhere has a marked tendency to become cystic. In one of the cases of glioma of the optic nerve that I reported[4] such a cyst was found on the disk [fig. 8].

In this case there was a glioma of the optic nerve, and ophthalmoscopy showed that the disk proper was replaced by a prepapillary nodule 5.5 by 4.5 mm. at the summit of which nodule was a cyst 2.5 mm. in diameter. This cyst was identical with the smaller cyst seen in the tumor proper and arose in the same way from cystoid degeneration of the neuroglia cells.

According to Durst, it was Hirschberg who mentioned a case of retinitis pigmentosa in a youth with good central vision, who showed in both fundi a cyst resembling a cysticercus, floating around in front of the optic disk.

It seems to me that in my patient I shall have to wait for definite neurologic signs of tuberous sclerosis before making that diagnosis and meanwhile content myself with the assumption that cysts of the optic disk can occur in cases of retinitis pigmentosa due to cystic changes taking place in areas of gliosis.

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4. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerve: A Histologic Study of Eleven Cases Including Case Showing Cystic Involvement of the Optic Disk, Tr. Sect. Ophth., A. M. A., 1921, p. 146; Arch. Ophth. **51**:120 (March) 1922, case 1.

ELEPHANTIASIS OF THE EYELID

M. N. BEIGELMAN, M.D.
LOS ANGELES

True elephantiasis of the eyelids is sufficiently rare to justify reports of new cases. Carletti,[1] in his recent monograph on this subject, stated that he could find in the literature only twenty-two cases of blepharo-elephantiasis, although the first report was published as far back as 1888.[2] This is a small number, particularly when one considers the serious deformity that is associated with elephantiasis and that easily attracts the observer's attention.

REPORT OF A CASE

Mr. G., 35 years of age, was first seen by me in November, 1928, in the Eye Clinic of the Eye and Ear Hospital of Los Angeles. According to the patient, his right eye was always in good condition. The left one was affected for many years by "trachoma," and about eight years before the present examination he noticed that the upper eyelid was gradually becoming thicker. During the last two years this eyelid became so large and heavy that he was unable to raise it (fig. 1), but there was no pain or tenderness.

On examination of the right eye, no pathologic changes were found. The upper eyelid of the left eye was enlarged to about twice its normal size. The skin appeared to be considerably thickened and less movable than normal, with no pitting on pressure. No circumscribed tumefaction could be palpated through the skin. Eversion of the upper lid, which was accomplished with some difficulty, revealed a generally hypertrophied palpebral conjunctiva, cicatricial in some areas. There was present a moderate pericorneal congestion; the cornea was diffusely opaque, with the upper two thirds of its surface covered by a pannus. The anterior chamber was of normal depth. The pupil was contracted, with the iris partly adherent to the anterior capsule of the lens. The fundus could not be seen. The vision of this eye was ability to count fingers at the distance of 1 foot (32.5 cm.).

Because of the advanced trachomatous condition, complicated by extensive corneal involvement, a tarsectomy was decided on. This was performed through a palpebral incision. At the time of the tarsectomy a piece of tissue, including the whole thickness of the eyelid, was removed for a pathologic examination. During the operation the unusual density of the subcutaneous tissue manifested itself in the difficulty with which the infiltration anthesia was carried out. On incision the pretarsal tissues were found to be greatly thickened; the usual structure of the orbicularis was indistinct; the muscle tissue was of a waxy appearance.

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1. Carletti, P.: Elefantiasi delle palpebre, Rome, Tipografia Ditta Ludovic Cecchini, 1924.

2. Pedraglia and Deutschmann: Chronisches Lidödem bei erisipelas-artiger Entzündung mit Tumorenbildung an der Lidrandern, Arch. f. Ophth. **34**:161, 1888.

The specimen removed during the operation was fixed in a dilute solution of formaldehyde and in Zenker's solution. Sections were stained for amyloid degeneration and for bacteria with negative results. For histologic studies hematoxylin-eosin, van Gieson, polymorphous methylene blue (methylthionine chloride, U. S.P.), orcein and Mallory's connective tissue stains were used. No changes of significance were detected in the epidermis. In the cutis the pars papillaris was hardly perceptible, while the reticular layer was greatly thickened. The latter consisted of large bands of connective fibers arranged partly in compact bundles and partly in a loose network (fig. 2). The upper part of the derma appeared to be edematous. The blood vessels were increased in number and enlarged. Prevalent throughout the cutis and the subcutaneous tissue was an infiltration of small cells, with irregular distribution. In some sections it was diffuse, while in others it formed sharply outlined follicles. It was commonly seen near the larger blood vessels, and it extended deeply into the muscular area. The infiltration cells were mostly of the lymphoid and plasmatic type. Comparatively few fibroblasts were present. Elastic tissue was scarce in sections stained with orcein.

Fig. 1.—Patient unable to raise the thickened left upper eyelid. Note the contraction of the frontal muscle on this side.

In the orbicular muscle the infiltration was less extensive than in the cutis and subcutaneous tissues, being limited to the interstitium. The muscle fibers, particularly in longitudinal sections (fig. 3), appeared partly structureless and converted into homogeneous hyaline material.

The tarsus (fig. 4) was little affected. It was generally thickened, and in some sections evidences of a round cell infiltration were found.

The conjunctiva (fig. 5) was hypertrophied. Immediately under the epithelial layer was a marked infiltration, consisting of lymphocytes, plasma cells, some mast cells and occasional fibroblasts.

DIAGNOSIS: SOFT TISSUE ROENTGENOGRAMS

In cases of diffusely enlarged eyelids, the diagnosis of blepharoelephantiasis is definitely established when histologic examination reveals an inflammatory fibromatosis of the derma and hypodermal tissues. In the absence of microscopic data the diagnosis of elephantiasis may become quite difficult. It is evident from a study of the literature pertaining to this subject that on many occasions cases of palpebral tumors (fibromas, lipomas, angiomas), leukemic infiltrations and even simple

edema were misdiagnosed and reported under the heading of elephantiasis of the eyelids. In the clinical differentiation between true blepharo-elephantiasis and conditions similar to it in appearance, the history of the case and the results of a physical examination may bring out some

Fig. 2.—Section of skin from the affected eyelid, showing diffuse inflammatory infiltration, some edema and newly formed connective tissue.

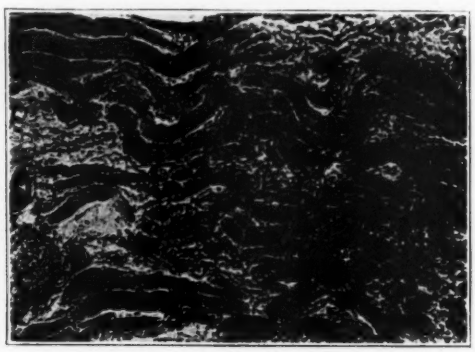

Fig. 3.—Longitudinal section of the orbicular muscle, showing interstitial infiltration and degenerative changes.

important diagnostic points. A tumefaction of the eyelid that has existed from birth is prone to be a congenital tumor rather than elephantiasis. Multiple fibromas on the body surface, a blood picture characteristic of leukemia and evidences of myxedema tend to detract from the diagnosis of blepharo-elephantiasis. Palpation of the affected

eye is of great value. Pitting on pressure is not encountered in elephantiasis; on the contrary, it is common in simple lymphedema. A circumscribed tumefaction suggests the presence of a tumor, while elephantiasis not only affects diffusely the whole eyelid, but frequently spreads to the adjoining part of the forehead, the temple, the nose and the face in general. The skin can often be raised in a fold over palpebral tumors; it is thickened and it cannot be folded in elephantiasis.

Fig. 4.—The tarsus, practically without change.

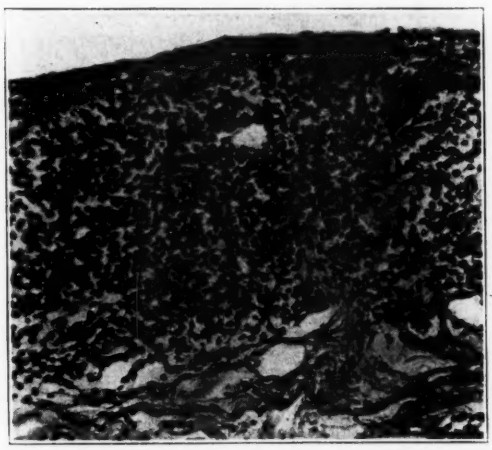

Fig. 5.—Inflammatory infiltration in the thickened tarsal conjunctiva.

In recent years so-called soft tissue roentgenograms have been introduced as a means of distinguishing pseudo-elephantoid conditions. According to F. L. Reichert,[3] these roentgenograms in cases of elephantiasis show an extensive network of trabeculations, representing advanced subcutaneous fibromatosis. Such trabeculations are never seen in simple lymphatic or vascular edema. It seems to me that the eyelids

3. Reichert, F. L.: The Recognition of Elephantiasis and of Elephantoid Conditions by Soft Tissue Roentgenography, Arch. Surg. **20**:543 (April) 1930.

are even more suitable for this roentgenographic study than are the extremities. It is indeed easy to obtain a bone-free roentgenogram of the palpebral tissues by inserting the x-ray film between the lid and the eyeball (Vogt). In obscure cases of palpebral tumefaction, particularly if a biopsy is not obtainable, this method may be of deciding diagnostic value.

PATHOLOGY

The two outstanding histopathologic features in the reported case were: (1) the inflammatory infiltration extending throughout the derma, subcutaneous tissue and orbicular muscle, and (2) the advanced fibromatosis portrayed by the large amount of newly formed connective tissue. The latter was represented chiefly by compact partly hyalinized bundles, and to a lesser degree by a loose and obviously edematous network.

There was no enlargement of the lymph spaces in any of the sections examined—a fact of some interest because of the prevailing opinion that distended lymph vessels are a necessary part in the histologic picture of elephantiasis. A rather emphatic statement to this effect can be found in a recent discussion on the histopathology of blepharoelephantiasis by Kümmel:[4] ". . . . am wichtigsten ist jedoch die Erweiterung und Neubildung der Lymphbahnen." (Most important, however, is the dilation and new formation of lymphatic pathways.) This particular stress laid on the microscopic evidences of lymphatic stasis is in accordance with the old theory of elephantiasis as the outcome of prolonged subcutaneous lymphedema. As will be shown in the discussion of the etiology of elephantiasis, a different factor—infection—predominates in the modern pathogenic conception of elephantiasis. Therefore, the absence of lymphatic derangement in many specimens examined histologically does not involve necessarily an assumption that previously dilated lymph spaces became obliterated in the process of extensive fibromatosis. It is interesting that in the newest and most thorough work on the pathology of elephantiasis by Alberca[5] in del Rio Hortega's laboratory, the lack of change in the lymphatic system is strongly emphasized.

Conflicting opinions exist as to the amount of elastic tissue in elephantiasis. This can be partly attributed to the variety of methods used in histologic examinations or to the degree of differentiation in the process of staining for elastic tissue fibers. As a rule, with the

4. Kümmel, R.: Lider, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1931, vol. 11, p. 196.

5. Alberca, R.: Contribution à la connaissance histopathologique de l'éléphantiasis nostras, Compt. rend. Soc. de biol. **2**:813, 1924.

progressive new formation of dense connective tissue, the elastic fibers decrease in numbers.

Pressure from the vastly increased amount of fibrous tissue, causing a partial disappearance of elastic fibers, is also responsible for the degeneration of the orbicularis in the reported case. The interstitial myositis might have contributed to the regressive changes in the muscle through interference with its proper nutrition.

ETIOLOGY: TRACHOMA AND BLEPHARO-ELEPHANTIASIS

Investigations repeatedly conducted in the last forty years have established beyond doubt the infectious origin of elephantiasis. The lymphatic stasis may precede the hyperplasia of connective tissue or be associated with it. But no matter how long the lymphedema exists, it cannot cause the elephantoid state unless an infection intervenes. On the contrary, "the progressive fibromatosis which we recognise as elephantiasis nostras, may occur independently of any stagnatory state of the lymphatic or venous circulation, and solely as a result of repeated attacks of streptococcal infection, which has been regarded by many as identical with the erysipelas coccus of Fehleisen" (Matas [6]). Erysipelas is by no means the only type of infection that may lead to elephantiasis. In fact, it was brought out recently by Kuntzen [7] that erysipelas is a secondary complication of the elephantoid state rather than the primary lesion. In Kuntzen's opinion the abnormal status of the skin in elephantiasis is responsible for frequent streptococcus invasion and attacks of erysipelas. According to Bertwistle and Gregg,[8] various infectious diseases, including tuberculosis, syphilis and leprosy, are known to cause elephantoid changes. It is of particular interest that among the chronic inflammations that result in elephantiasis Kümmel considered trachoma, quoting two cases of this kind reported by Polignani [9] and de Vincentiis.[10] The unmistakable signs of trachoma on the side of blepharo-elephantiasis in the present case have prompted me to investigate more closely the possible relationship between trachoma and elephantiasis of the eyelid. A study of Polignani's and de Vincentiis'

6. Matas, R.: The Surgical Treatment of Elephantiasis and Elephantoid States Dependent upon Chronic Obstruction of the Lymphatic and Venous Channels, Am. J. Trop. Dis. **1**:60, 1913.

7. Kuntzen, H.: Die Chirurgie der Elephantiasis, Arch. f. klin. Chir. **158**:543, 1930.

8. Bertwistle, A. P., and Gregg, A. L.: Elephantiasis, Brit. J. Surg. **16**:267, 1928.

9. Polignani: Elefantiasi linfangioide parziale delle palpebre, Lavori clin. ocul., Napoli **3**:181, 1893, quoted by Carletti (footnote 1).

10. de Vincentiis: Elefantiasi neuromatode della palpebra superiore destra, Lavori clin. ocul., Napoli **5**:66, 1893, quoted by Carletti (footnote 1).

contributions reveals that these authors were dealing with tumors and not with true elephantiasis of the eyelids. Polignani's case must be classified as one of lymphangioma, which as a matter of fact was subconjunctival and invaded neither the derma nor the subcutaneous tissues. In de Vincentiis' case a histologic examination brought out the characteristic picture of a plexiform fibroma. I am therefore justified in stating that previous to the present observation there was no case on record of combined blepharo-elephantiasis and trachoma. Considering the enormous amount of trachoma material and the conspicuousness of elephantoid conditions, this total lack of reports on the simultaneous occurrence of both diseases is in itself significant. An attempt to establish the relationship between trachoma and blepharo-elephantiasis on the basis of the microscopic observations in the present case meets with serious difficulties. There is no histologic picture pathognomonic of trachoma, and therefore no conclusion can be drawn from the morphology of the inflammatory changes in the reported case. There is also little known, generally, regarding the depth and extent of a trachomatous infiltration, and it is still an open question whether it ever involves the orbicular muscle and the subcutaneous tissues of the eyelids. Microscopic study of the affected eyelid brought out only one fact of importance, namely, that the tarsus formed a sort of barrier between the two infiltration areas—in the conjunctiva and in the subcutaneous tissues of the eyelid. The intensity of the inflammatory process was definitely decreasing from both the surface of the skin and the conjunctiva in the direction of the tarsus, suggesting that the two diseased processes were independent of each other.

It therefore appears that, at the present state of the knowledge, there are reasons to consider the concurrence of trachoma and elephantiasis as a rare coincidence, without any causative interrelation.

Since 1924, when A. Vogt [11] for the first time reported an enlargement of the sella turcica in unilateral blepharo-elephantiasis, considerable attention has been given to this fact. Metzger,[12] Mintschewa [13] and Awguschewitsch [14] contributed similar observations. The possible rôle of hypophyseal dysfunction in the origin of blepharo-elephantiasis

11. Vogt, A.: Einseitige Elephantiasis des Oberlides bei erweiterter Sella turcica, Klin. Monatsbl. f. Augenh. **72**:507, 1924.

12. Metzger: Elephantiasis der Lider und Hydrophthalmus mit Erweiterung der Sella turcica, Klin. Monatsbl. f. Augenh. **75**:248, 1925.

13. Mintschewa, Minka: Erweiterung der Sella turcica bei einseitiger Elephantiasis der Lider und Hydrophthalmus infolge Recklinghausenscher Krankheit, Klin. Monatsbl. f. Augenh. **76**:403, 1926.

14. Awguschewitsch, P. L.: Elephantiasis des oberen Augenlides, Hemihypertrophia faciei und Hydrophthalmus mit gleichzeitiger Erweiterung der Sella turcica, Klin. Monatsbl. f. Augenh. **83**:91, 1929.

became a matter of some speculation, although in the only case of blepharo-elephantiasis with enlarged sella turcica that came to section no changes in the hypophysis were found. Schinz stated that "those suffering from elephantiasis of the upper eyelid should be subjected to an x-ray study of the sella turcica." This study was made in the present case with negative results: The sella turcica was normal in size and shape.

TREATMENT

There is no doubt that attention to constitutional disorders, removal of septic foci and similar measures offer the patient a better chance to combat the infectious process that is at the root of elephantiasis of the eyelids. In favorable instances, a sufficiently improved resistance may bring to an end the inflammatory activities, and with them the progressive enlargement of the eyelid. It is obvious, however, that the fibrous tissue already formed can be removed only by some surgical procedure. Cirincione [15] accomplished this through an incision along the eyebrows, with a subsequent undermining of the skin. The excessive fibrous tissue together with a part of the orbicular muscle were then removed. In the present case the enlargement was almost entirely confined to the lower half of the upper eyelid. I thought it more advantageous to remove from this lower part of the eyelid an oval piece of tissue, including all its layers from the epidermis to the conjunctiva, care being taken not to damage the levator. The edges of the wound were then carefully approximated both anteriorly and posteriorly. The outcome of this procedure was quite satisfactory: The patient could keep his eye open, and the general appearance of the eyelid was markedly improved. This result remains unchanged fourteen months after the operation.

15. Cirincione: Elefantiasi delle palpebre, Clin. ocul. **1**:69, 1900, quoted by Carletti (footnote 1).

METASTATIC CARCINOMA OF THE OPTIC DISK

WITH REPORT OF A CASE

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WASHINGTON, D. C.

According to statistics, it is estimated that 50 per cent of all cancers metastasize and that 4.77 per cent involve the central nervous system.

I have been unable to ascertain if the percentage of metastases to the eye has been determined when the central nervous system is involved; it would be of interest to know.

Winkleman and Eckle [1] have determined that about one half of the metastases of the central nervous system arise from primary cancers of the breast, lung and prostate, with the uterus next on the list of frequency.

When the metastasis is by way of the lymphatics, the meninges are more apt to be involved.

Retrograde extension, first emphasized by von Recklinghausen and later confirmed by others, is a frequent means of extension of cancer, and occurs more especially in organs having a venous pulse.

Ewing thinks that the reason some organs are involved more frequently than others is in the circulatory condition of these organs.

Metastasis to the central nervous system may occur at any time, even years after the appearance of the cancer or after the operation on the primary tumor, or it may occur before the primary growth is suspected.

In those with symptoms of tumor of the brain or with symptoms of involvement of this organ in a diseased process, one should bear in mind the possibility of a metastasis from an as yet unrecognized cancer of the prostate, lung or breast. Naturally there is no group of symptoms in any way characteristic of involvement of the brain, since the growth may be disseminated to the several parts of this organ. The surgeon can imagine his dismay, on opening the skull to determine the location of a supposedly operable tumor, to find that he has to deal with an infiltrating cancerous growth.

Grinker [2] cited a case of glioma involving the chiasm extending along the optic nerve. The right disk showed choking in its upper

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Read before the Section on Ophthalmology at the Eighty-Third Annual Session of the American Medical Association, New Orleans, May 13, 1932.

1. Winkleman, W. W., and Eckle, J. L.: J. Nerv. & Ment. Dis. **66**:1 (July); 133 (Aug.) 1927.

2. Grinker, R. R.: Tumors of the Optic Nerve, Arch. Ophth. **4**:497 (Oct.) 1930.

half and paleness of its lower half. This involvement of half the disk is analogous to the case of carcinoma of the disk to be reported.

Clapp [3] reported 118 cases of metastatic carcinoma of the choroid. He described the lung as the second commonest primary focus for such metastases, the breast being the usual focus.

Involvement of the intra-ocular end of the optic nerve is rare, and, so far as a careful search can determine, involvement of part of the disk, with choking of a portion of it, has been reported in the literature in but three cases. In one it was the extension of a glioma, as already mentioned. H. Knapp and von Graefe each mentioned a case of carcinoma with choking of part of the disk.

Knapp [4] reported as follows: The tumor was a scirrhous cancer surrounding the optic nerve posterior to the globe: it was enucleated, with preservation of the globe. When first seen, the disk was swollen, with abrupt edges. The arteries and veins were not visible on the swollen disk, which was grayish white. The pseudoplasm did not involve the nerve, but only the sheath. These changes were evidently propagated into the end of the optic nerve where they produced a descending neuritis. The elevation of the intra-ocular end of the optic nerve, one may fairly assume, resulted from the presence of the same round cells that were found so abundantly infiltrated in the nerve, as well as from the pressure on the blood vessels of the optic nerve, causing serous exudation into the tissue. The absence of any growth in the fundus showed that there was no extension of an intra-ocular tumor into the optic nerve. The preservation of vision and a complete visual field made it probable that the growth did not originate in the nerve or penetrate deeply into it, but rather that it sprang from the tissues in the subvaginal space or from the outer sheath. The abrupt elevation of the disk and the limitation of the swelling were characteristic and were thought to be due to infiltration of the disk with cells and their derivatives. A serous elevation would produce a swelling of the disk with more sloping borders.

As will be seen, this was characteristic of the swelling of the disk face in my case, which I took to be due to the infiltration of the nasal side of the disk as a result of extension from the sheath of the nerve on this side.

Knapp went on to say that a steep and limited choked disk might refer to a proliferating extra-ocular process.

Von Graefe also mentioned the steep elevation of the papilla in his case. Limitation of the visual field has been observed in cases in

3. Clapp, C. A.: Am. J. Ophth. **9**:513, 1926.

4. Knapp, H.: A Case of Carcinoma of the Outer Sheath of the Optic Nerve Removed with Preservation of the Eyeball. Arch. Ophth. & Otol. **4**:323, 1874-1875.

which the neoplasm originated in the inner sheath and destroyed a part of the nerve.

A great number of cases of carcinomatous involvement of the extra-ocular portion of the optic nerve have been reported; they have been metastatic or by extension along the nerve.

It is the cases in which the intra-ocular end of the nerve is involved that are rare and in which the disk presents such a startling appearance.

Von Haselberg [5] remarked that metastatic carcinoma occurs in many of the young, particularly in women with cancer of the genital tract or mammary gland. One or both eyes may be involved. If one eye becomes involved, it is more frequently the left, since it is along the most direct route of the blood channels, the left common carotid artery

Fig. 1.—Fundus (left eye) on June 15, 1930.

branching off directly from the arch of the aorta. Mammary carcinoma in the young particularly shows a marked capacity for dissemination. It is certain that far more metastases occur in the eye than one assumes. One is rarely in a position to determine ocular disturbances in patients dying of carcinoma cachexia. Semiconscious patients are not aware of a decrease of their vision. They may not notice that they have lost the vision of one eye.

REPORT OF A CASE

A married woman, 31 years of age, presented herself in June, 1930, complaining of severe headaches which began six months before I saw her. In June, 1929, she was operated on for scirrhous cancer of the right breast by Dr. Charles S. White.

5. von Haselberg, W.: Metastatic Carcinoma of the Optic Nerve, Berl. klin. Wchnschr. **51**:1237, 1914.

who gave me his report. By frozen sectioning a diagnosis of scirrhous carcinoma was made and a radical operation done. The lymph nodes of the axillary region were involved. The activity of the carcinoma cells was more pronounced in the metastatic region than in the breast. Dr. Hunter McGuire saw this patient in May, 1930; he gave me the following report on the ocular condition at that time: "In the left eye there was a large elevated whitish area overlapping about half the diameter of the disc and extending several disc diameters into the adjacent fundus. This was best seen with a +6 lens. Transillumination was negative. The right eye was negative except for an old area of choroiditis below the macula and a few pigmentary degenerations in the extreme nasal periphery." This patient was carefully studied by Dr. Clark in June, 1930, and the following diagnoses were made: chronic cholecystitis; chronic constipation; psychoneurosis, anxiety type; secondary anemia; a malignant growth, an early recurrence; laceration of the cervix with erosion.

Fig. 2.—Fundus in September, 1930.

This patient informed me that her headaches were growing progressively worse and that of late they had been attended with vomiting. Her vision was 20/15 in each eye, with a moderate myopic correction. There was some involvement of the peripheral fields but not of the central fields. The left blind spot was definitely enlarged.

The patient was not in the best of condition for such an examination, since she was somewhat bewildered and suffering from excruciating headache. It was obvious that cerebral metastasis had occurred. The right pupil measured 3 mm. and the left 4 mm., both reacting normally to light and to accommodation. Extraocular muscle balance was normal.

The ophthalmoscopic findings were as follows: In the right eye, the disk and retinal vessels were normal. In an area beginning ¾ disk-diameter to the temporal side of the disk, bounded approximately by the superior branch of the superior temporal artery above and by a line on the plane of the macula below, and extending from 8 to 10 disk-diameters temporally, the retina was grayish white. It was

stippled with a number of small black pigmented dots and larger pigmented areas, and was 1 diopter above the level of the adjacent retina.

About the edge of the macula there were a number of glittering shining reflexes, giving the appearance of a macula star. They were undoubtedly reflexes, since they disappeared with the shifting of the light. One sees such changes, however, in the retina that are not normal and that are approaching pathologic conditions demonstrable ophthalmoscopically.

In the left eye, the temporal half of the disk was normal. The nasal half was elevated 6 diopters. This portion of the disk was separated from the normal

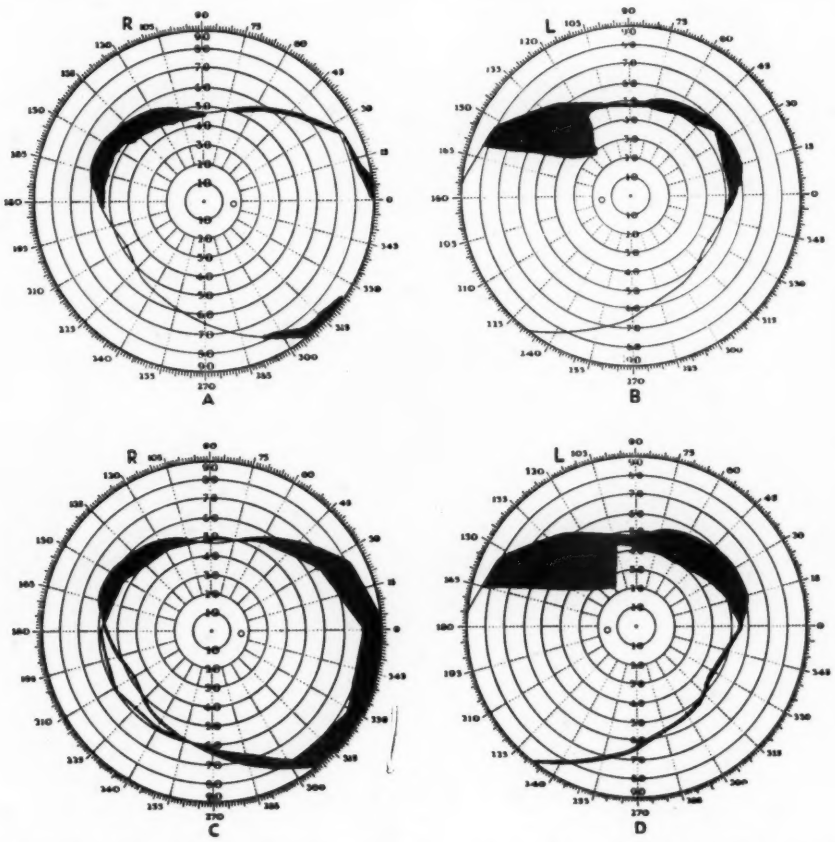

Fig. 3.—Visual fields in case reported: *A*, *B*, on June 17, 1930, 6/330. *C*, *D*, on September 15, 3/330.

temporal half sharply, so that there was a perpendicular wall dividing the two halves, presenting a most unusual appearance. This elevation extended into the retina above the disk 3 disk-diameters, gradually sloping off into the normal retinal level. Nasally it extended 8 disk-diameters, also gradually sloping to the normal retinal level; below it extended 4 disk-diameters, dropping off rather sharply to the retinal level.

The surface of this elevation was grayish white. There was no movement or undulation over it. It was indistinctly lobulated. The vessels over this area were normal in appearance. Transillumination was unsatisfactory and gave no informa-

tion, since the growth was at the posterior pole of the globe. It was not thought wise to use posterior transillumination. Oral transillumination did not show any difference in the pupillary glare of the two eyes.

It was thought at the time of examination that this elevation of the disk and retina was due to an intracranial metastatic carcinoma that extended along the sheath of the optic nerve without involving the nerve itself to any extent, as indicated by the visual fields. It probably extended along the sheath on the nasal side and infiltrated the choroid in this region.

Figure 1 illustrates the appearance of the disk and retina in its neighborhood as described. This was made June 15, 1930.

The patient was again seen on September 15. The right fundus, in the area previously described, presented a grayish-white appearance, with a number of small black pigmented dots and larger black ones scattered over it. The level of this area was 1 diopter above that of the normal retina. The same star figure

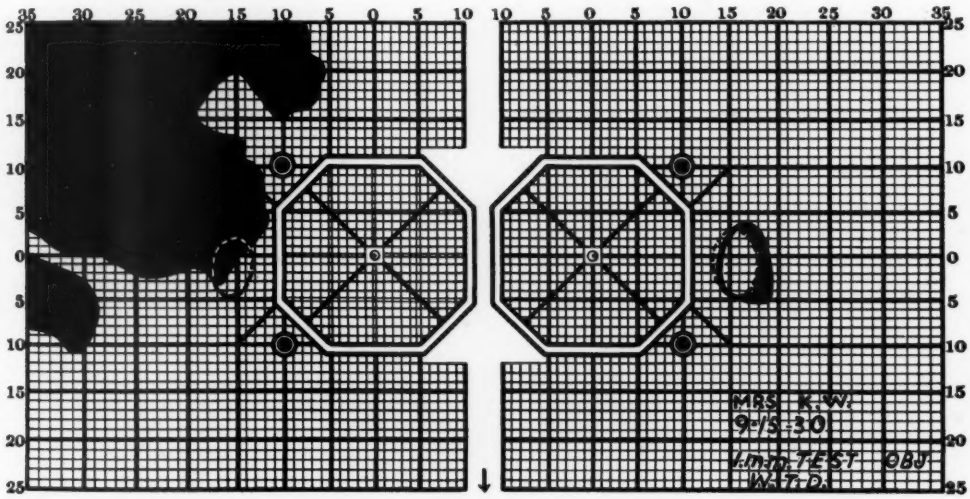

Fig. 4.—Stereo-campimetric fields obtained on September 15, using 1 mm. test object.

appearance of the reflexes was present at the macula. In the left fundus one third of the disk was normal on the temporal side; the margin of the disk adjoining this area was sharply outlined. There was moderate choroidal disturbance at the margin. At the lower margin at 5 o'clock there was a sudden elevation of the tissues of the disk in the form of a vertical wall dividing the normal from the choked part of the disk. This elevation rises to 6 diopters at its highest point. Above, this elevation was slightly less abrupt and occurred at 12:30 on the disk. The central portion of the elevation spread over one half of the physiologic cup, the temporal half of the cup being normal. The highest point of the elevation was opposite the temporal edge of the disk. The outlines of the elevation were as first described.

The following vessels were involved in the elevated area: the inferior temporal artery and vein; the inferior nasal artery and vein; the superior nasal artery at its beginning; the nasal branch of the superior nasal artery and vein.

At the junction of the elevated portion of the retina with the normal portion there were a number of silvery white spots and also a number of horizontal striae,

the latter probably formed by folds in the retina. The surface of the elevation presented a mottled appearance. There was no evidence of further change in the retina. The appearance of the fundus is shown in figure 2 made in September.

The question as to whether this was a beginning choroidal metastasis arises in one's mind; if so, its progress was slow. I would be inclined to attribute the ophthalmoscopic appearances to involvement of the choroid in a metastatic cancerous process.

The patient died in November, 1930. I quote from a letter from her husband: "She seemed to lapse into a semicoma and did not seem to suffer greatly. She did not lose her eyesight." It seems evident that the cerebral growth was the cause of death. Every possible effort was made to obtain an autopsy, without success. It is unfortunate that laboratory proof is lacking, but one is justified, I think, in reporting the case clinically as carcinoma of the intra-ocular portion of the optic nerve.

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ABSTRACT OF DISCUSSION

DR. GEORGE FRANCIS SUKER, Chicago: It is unfortunate that an autopsy was unattainable in this rather unusual case. The accurate clinical description of the mammary carcinoma, the detailed observations on the fundus and death following shortly after the metastasis leave no room for doubt as to the correct diagnosis, namely, metastatic carcinoma of the nervus opticus papillae. Much rarer than a carcinomatous metastasis of the optic nerve, sheath or disk is a metastatic sarcoma of these parts, and even the ocular carcinomatous metastasis is unusual.

Carcinomatous metastasis occurs about once in 30,000 cases, according to Uhthoff, once in 150,000 cases according to Sattler, and in my own experience, once in about 100,000 cases. There were on record, up to 1909, 64 cases of authenticated metastatic intra-ocular carcinoma (Suker and Grosvenor: *Am. J. Ophth.* **5**:580, 1909), and Clapp (ibid. **9**:153, 1926) added an additional 54 up to the year 1926. These statistics started with the year 1872.

Authentic cases of metastatic carcinoma of the eye date from 1872, when Perls found a choroidal metastasis in a man of 43, the primary carcinoma having been in the lung. Ten years later Hirshberg was the first to describe a bilateral metastatic carcinoma of the choroid in a woman, aged 52, who for nine years suffered from a mammary carcinoma. Schoeler also described a case in 1882.

In more than a third of all cases both eyes were involved — seldom simultaneously — but the second eye is apt to be involved shortly after the first. The left is more often the seat of the metastasis than the right, because the left internal carotid arises directly from the aorta, while the right internal leaves the innominate at an obtuse angle. For this reason also, emboli of the brain are more frequent in the left than the right hemisphere.

That carcinoma metastases to the orbit or optic nerve are a clinical rarity is because the ophthalmic artery leaves the internal carotid at a right angle, allowing the carcinoma cells or emboli in the vessel readily to pass the rather narrow ostium of the ophthalmic artery; they are then carried on to the brain or meninges.

These metastases usually occur about two years after the detection of the primary carcinoma. After the metastasis, death occurs usually within nine months to one year. This was my observation from the study of the 64 cases that I reported in 1912. However, death frequently occurs within but a few months.

Of all the anatomic structures of the eye, the choroid is the one part that is usually the seat of metastatic carcinoma, and then in the region of its posterior pole, seldom above or below the entrance of the optic nerve or on its nasal side (Sattler). This is easily understood because of the more than 20 short ciliary arteries in this locality, while only 2 long ciliary arteries travel to the iris and there is only 1 central artery of the retina.

Unlike sarcoma, carcinoma does not proceed in the preformed passages of the various ocular structures, i. e., scleral vessels and nerves. For this reason epibulbar metastatic carcinoma is decidedly less frequent than epibulbar metastatic sarcoma.

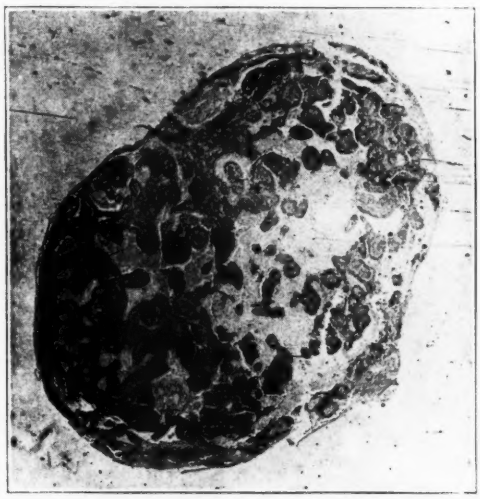

Fig. 5.—The circumference of nerve was about two and one-half times that of a normal nerve.

In some instances the choroidal carcinoma passes into the optic disk, and thence can proceed into the nerve proper, which in the case of Dr. Davis may have been the course. Frequently there are several metastatic nodes. From the disk the tumor can invade the retina, as in cases cited by Oatmon (*Am. J. M. Sc.*, March, 1903), Steichle (*Arch. f. Augenh.*, 1919, vol. 84), Lagrange (*Arch. d'opht.*, 1898, vol. 18) Oeller (*Arch. f. Augenh.*, 1905, vol. 52) and Gelpke (*Klin. Monatsbl. f. Augenh.*, 1905, vol. 43).

In some instances there are retrobulbar metastases which incorporate or envelop the optic nerve, as in the cases reported by Schoeller (*Centralbl. f. prakt. Augenh.* **7**:236, 1883) and Uhthoff (*Med. Festschr. f. Virchow* **5**:11, 1891). In this connection I cite a case of carcinoma of the epipharynx (with the permission of Dr. Howard C. Ballenger, Chicago): A man, aged 40, had primary carcinoma of the cervical glands, on the right and left sides, in 1927. The glands, on removal, showed carcinoma of the transitional cell type. The original focus or tumor was found in Rosenmüller's fossa near the orifice of the eustachian tube.

At autopsy, an infiltrating tumor mass was found to occupy a large area in the left middle fossa. The dura mater in the medial half of middle fossa was largely replaced, by firm tumor tissue; it invaded the left temporal cerebral lobe, the posterior half of the left frontal lobe, the left side of the pons and the left optic tract and chiasm. The left optic and oculomotor nerves were completely surrounded and apparently replaced by a tumor mass. The growth extended into the left optic foramen and filled the posterior third of the orbit with soft, friable pink tissue.

Fig. 6.—Marked tumor infiltration and destruction of nerve fibers.

The microscopic diagnosis was transitional cell carcinoma, arising from the nasopharyngeal mucosa, with extensive infiltrations of the base of the skull and brain.

A similar case was reported by Cords (*Versamml. d. deutsch. ophth. Gesellsch.*, 1922) which showed a metastasis of the meninges involving the optic sheaths and extending up to the cribriform plate, also penetrating the optic septums, but leaving some of the nerve fibers intact. The primary tumor was a gastric carcinoma.

These intra-ocular and nerve metastases usually arise from a carcinomatous breast, uterus, lung, brain or prostate, in the order of frequency mentioned.

Furthermore, when there is an intra-ocular metastasis other distant organs are also implicated.

The prognosis of any case of metastatic carcinoma is serious and eventually fatal. If a metastasis does take place, more appear in other organs than in the organ showing the first metastasis. Intra-ocular metastasis is the most serious of any, as death usually occurs within a month to one year from the time of the appearance of the ocular metastasis. For this cogent reason, enucleation should never be done unless the tumor within the globe is of rapid growth, perforates or causes such pain (glaucoma) as to necessitate relief.

Fig. 7.—Magnification of figure 6.

Carcinomatous metastasis to the optic nerve or its sheaths is a rare clinical finding. Goldstein and Wexler (Metastasis in Sheath of Optic Nerve from Carcinoma of Stomach, ARCH. OPHTH. **6**:414 [Sept.] 1931, with bibliography) reported metastasis to the optic sheath. In this case vision was not destroyed in either eye. The uvea and disk were normal, as was the optic nerve itself. Goldstein and Wexler found only 4 similar cases in the literature; the case reported here makes the fifth. However, in none of the four cases was the optic nerve itself so completely destroyed and replaced by tumor tissue within the sheath as in this briefly reported case.

The difference between these 5 cases and the one that Dr. Davis cited is that the former are retrobulbar nerve metastases, while Dr. Davis' case is a metastasis from the choroid into the disk and then along the nerve.

Metastatic carcinoma within the eyeball and the optic nerve up to the chiasm are not so unusual as the statistics of Clapp, Suker and Grosvenor would indicate, for the following reasons:

1. Intra-ocular metastasis often appears late, frequently when the patient is already more or less moribund.

2. The intra-ocular metastasis is often very small and in such sections of the fundus as not to cause more visual disturbance, as the patient is frequently in a state of carcinomatous cachexia.

3. The metastasis in the choroid is frequently of slower growth than metastasis in the brain or elsewhere, and the patient succumbs before attention is attracted to the eye.

4. Only too infrequently is the eyeball included in the routine postmortem examination. Many globes taken from patients who have died of carcinoma have shown choroidal carcinoma.

It is because intra-ocular metastasis usually occurs after the primary carcinoma has been operated on, and many other organs already show metastasis that the ocular metastasis is overlooked or neglected.

5. Then, too, ophthalmoscopic examination is not a routine procedure in the general physical examination in this class of cases.

Dr. W. L. Benedict, Rochester, Minn.: Metastatic adenocarcinoma appears about the optic nerve head, but is located more often in the choroid near the disk and produces an ophthalmoscopic appearance of swelling of the nerve. The tumor may extend from the choroid into the optic nerve. I wish to report two cases and to show sections of metastatic carcinoma of the choroid near the optic disk.

Case 1.—Mrs. H., aged 44, had primary carcinoma of the left breast removed in February, 1926. She came to the clinic on June 4, complaining of failing vision in the left eye. At the time of her operation in February, 1926, she had noticed disturbance of vision in the left eye, as though she were seeing heat waves. During the three weeks previous to her visit to the clinic she had noticed flashes of light and colored spots. From May 24 to June 1 there was considerable pain in the left eye, partially relieved by acetylsalicylic acid. She noticed disturbances in the field of vision of the left eye which persisted. Vision of the left eye was 6/5-3. Ophthalmoscopic examination showed the media to be clear. There was a detachment of the nasal half of the retina extending from the disk forward to the ciliary body. The detached retina was gray, except near the central portion, where there was an elevated red mass over which the retina was not detached. The mass apparently arose in the choroid and extended over the globe. The eye was enucleated immediately. The diagnosis was grade IV metastatic carcinoma. The patient also showed metastases in the skin. A specimen of the tumor of the breast could not be obtained. A section from the eye showed large metastatic adenocarcinoma of the choroid, on the nasal side of the disk, pushing the nerve head far to the temporal side. The nerve was not invaded by the tumor.

Case 2.—Miss R., aged 48, came to the clinic in October, 1930, complaining of constipation and diarrhea. Proctoscopic examination showed annular carcinoma of the upper portion of the rectum. On November 17, 1930, a left inguinal colostomy was done. Abdominal metastases were not found. There was a small, freely movable mass in the rectosigmoid, just above the peritoneal fold. A biopsy

on the tumor showed grade I adenocarcinoma. On November 14, the vision of the right eye was 6/5, and of the left eye 6/7. The visual field was markedly restricted in the upper temporal and upper nasal quadrant. Ophthalmoscopic examination showed edema of the disk with detachment of the retina in the lower nasal quadrant. The eye was observed daily, and the mass could be seen to increase in size. The eye was enucleated on December 1. The microscopic section showed metastatic adenocarcinoma of the choroid entirely surrounding and covering the nerve head. The carcinoma was beginning to invade the disk, but did not extend beyond the lamina cribrosa.

WOUND CLOSURE FOR INTRACAPSULAR EXTRACTION OF CATARACT

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Among the great advances made in ophthalmic surgery in the recent past must be listed the general acceptance of some form of suture for closing the incision made for the extraction of cataract. Of great assistance in capsulotomy procedures, suture becames an absolute necessity in closing the larger incision required for intracapsular methods.

Since the first attempt made by Williams [1] in 1869 to suture a cataract incision, many methods of suturing have been advocated. These can be divided into several types as follows: (1) conjunctival, (2) corneal, (3) scleroconjunctival, (4) sclerocorneal and (5) sclerocorneal-conjunctival.

It is obvious that a purely conjunctival suture placed across the incision can give only partial security because of the elasticity of the conjunctiva and its friability, especially in the aged. A sliding flap of the Van Lint type anchored to the episcleral or outer scleral tissue is much more secure, but still does not prevent gaping of the wound under the flap.

A corneal suture requires the incision to be made entirely in corneal tissue, which is certainly not the best site for it.

The scleroconjunctival suture, as proposed by Verhoeff [2] in 1916 and later modified by Derby,[3] gives a secure closure of the wound at its vertex. The chief disadvantage of Derby's modification, as pointed out by Verhoeff,[4] is that pulling the suture too tight may cause overriding of the edges of the incision if it is not placed with accuracy.

Verhoeff's most recent suture [5] possesses many advantages over the other types mentioned. Since it passes through corneal and scleral tissues in the same radial line, no lateral displacement or distortion of the section can occur. Being anchored in the nonelastic tissues of the cornea and sclera, it provides greater security against gaping of the

1. Williams, W. H.: Arch. Ophth. & Otol. **1**:102, 1869.
2. Verhoeff, F. H.: Arch. Ophth. **45**:479 (Sept.) 1916.
3. Derby, G. S.: Am. J. Ophth. **8**:381 (May) 1925.
4. Verhoeff, F. H.: Am. J. Ophth. **8**:886 (Nov.) 1925.
5. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **25**:48, 1927.

wound than do any of the others. Because of the narrow gap left for the incision, more accurate closure is possible. It is possible to close the incision rapidly and thus control vitreous prolapse promptly and efficiently.

These various types of sutures, with the exception of the last, have been admirably discussed by Ellett.[6] Howard's [7] discussion of the value of sutures, especially of Derby's modification of the scleroconjunctival suture, is an important and valuable contribution. Verhoeff [8] and Derby [3] have published detailed descriptions of their sutures.

It is not our intention to present a new suture or to elaborate unduly on the subject, but to present two combinations of sutures, which have been of great value and which meet certain definite indications.

These indications are:

1. The incision must be closed securely and accurately. This is the most important requirement for preventing postoperative vitreous prolapse and for securing rapid healing.

2. Closure must be rapid. One should be able to close the incision within from five to fifteen seconds after the lens is delivered. This is important if vitreous presents behind the lens. We feel that this is the only point in the operation where speed is absolutely essential.

3. The suture must be placed before the incision is made, and therefore must be made in such a way as to allow passage of the knife, yet without too wide a gap, and at the same time to permit the incision to open wide enough for the insertion of an erisiphake or forceps and for the exit of the lens. A large incision is the first requisite of a successful intracapsular extraction. Placing the suture before making the incision enables the surgeon to be prepared for the prolapse or loss of vitreous, which, it must be admitted, is bound to occur in some cases, especially if an intracapsular technic is used. Loss of a small amount of vitreous, while not to be desired, is not a serious accident if the entire capsule of the lens is removed and the wound can be immediately and securely closed. Placing sutures while vitreous is oozing or presenting can result only in further loss of vitreous.

4. The conjunctival flap should be large enough to cover the entire incision, giving a truly surgical closure, tending to prevent infection of the wound and covering any small iris prolapse as soon as it occurs. No single suture can absolutely insure against prolapse.

5. Secure closure must be possible if the cataract is extracted within a few weeks after preliminary iridectomy.

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6. Ellett, E. C., in Contributions to Ophthalmic Science, Menasha, Wis., George Banta Publishing Company, 1926, p. 229.

7. Howard, H. J.: Tr. Sect. Ophth., A. M. A., 1924, p. 208.

8. Verhoeff (footnotes 2, 4 and 5).

The first three indications are met by the Verhoeff corneal-sclerocon-junctival suture. The Van Lint sliding conjunctival flap can be made in such a way as to cover the entire incision. The last requirement is met by Derby's modification of Verhoeff's original scleroconjunctival suture.

AUTHORS' METHODS

Our procedure is to prepare the eye as for the Verhoeff suture, but the conjunctival flap is enlarged around the limbus to the expected limits of the corneal section and dissected free as for a Van Lint sliding flap. The Verhoeff suture is then placed in the cornea, sclera and conjunctiva, and the loops are laid back so as not to interfere with the knife (fig. 1). The suture emerges through the conjunctiva somewhat farther back from its free edge than Verhoeff recommends, in

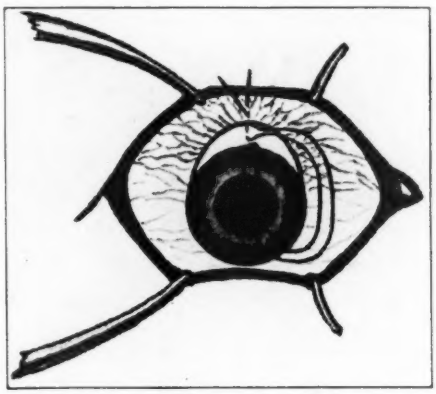

Fig. 1.—Van Lint flap dissected and Verhoeff suture in place.

order that the flap may be drawn down over the cornea a little more. In making the section, the knife emerges at the limbus in the gap between the loops of the suture. This gap needs to be only 1 or 1.5 mm. wide. After the lens is extracted, the suture, the ends of which have already been loosely tied, is tightened and tied by one assistant as the other grasps the flap with forceps and draws it down over the cornea. This can be done by the surgeon and one assistant.

The eye is then closed for ten minutes to allow the vitreous to settle back and to permit the partial reformation of the anterior chamber. The iris is then smoothed out. Since only the center of the flap has been pulled down, the sides still being retracted, a spatula can be passed into the anterior chamber on either side of the suture just as if no flap had been made. Following this, as the final step before dressing the eye, a suture is passed on either side through the conjunctiva below and slightly lateral to the angle of the incision. Each suture is passed through the edge of the flap at such a position that when tied they will

bring the flap down over the cornea, completely covering the incision, without making too much pressure on the cornea.

The second procedure is applicable in cases in which a preliminary iridectomy has been done a short time before the extraction. In such cases, the insertion of a suture into the cornea from above downward is likely to reopen the keratome incision, evacuating the anterior chamber, which makes the incision difficult. To obviate this complication, the dissection of the Van Lint flap is not begun at the upper limbus but behind the original keratome incision, leaving a semilunar flap the angles of which extend beyond the keratome incision on either side. This is carefully dissected downward to the cornea, and a scleroconjunctival suture is placed and looped back according to Derby's technic, except that the conjunctival flap has a broader base (fig. 2). The original Verhoeff scleroconjunctival suture is not well adapted for use

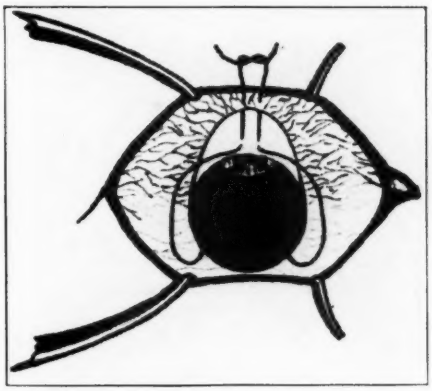

Fig. 2.—Van Lint flap dissected and scleroconjunctival suture in place. (Preliminary iridectomy.)

in combination with the Van Lint flap. The suture is placed in the flap close to the limbus and passed through the outer scleral fibers immediately behind the keratome incision, emerging well back in the large flap, so as to allow it to come down over the cataract incision. The Van Lint flap is dissected and sutured exactly as previously described. Figure 3 shows the appearance of the eye after the completion of either procedure.

In our first trials of this type of closure, we placed the central suture and then the lateral flap sutures before making the incision, anchoring the latter in episcleral or scleral tissue near the limbus. We soon abandoned this method because of the impossibility of placing the sutures in the flap so that it would be drawn down evenly on both sides of the central suture, after the latter was tied. Also, the extra sutures were very much in the way and were likely to be cut by the knife or interfere in other ways.

In suturing the Van Lint flap, no effort is made at fixation of the sutures in scleral tissue, in order to avoid undue pressure on the globe. The suture is passed through the pinched-up conjunctiva, allowance being made for a certain amount of stretching. No dependence is placed on these sutures for security; the flap merely covers the incision, the central suture providing the firm closure.

The conjunctival sutures are removed on the fourth or fifth day. The central suture is removed on the eighth day according to Verhoeff's practice, provided, however, that it is not a source of great irritation or that the flap has not retracted so as to uncover it, in which case it may be removed on the fourth or fifth day.

In removing the sutures, the eye is cocainized and the conjunctiva about the sutures is touched with a swab dipped in a solution of 20 per cent cocaine containing zinc sulphate, ¼ grain (0.016 Gm.) to the

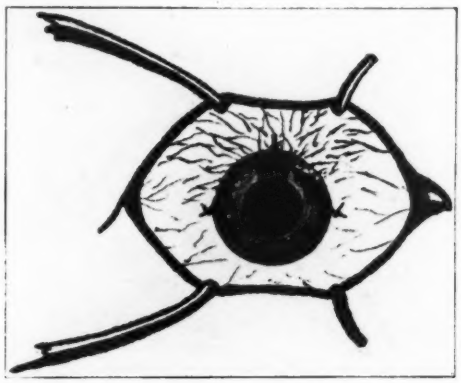

Fig. 3.—Final appearance of the eye. (The flap should come down slightly farther than is shown.)

ounce. The sutures are cut before being grasped with forceps to avoid hemorrhage and pain if the patient moves his eye suddenly. In removing the Verhoeff suture, it should be cut as close to the cornea as possible so that none of the suture material that has been free in the conjunctival sac will be dragged through the cornea.

The advantages of this form of closure of the wound are: 1. The Verhoeff suture provides firm, accurate closure of the wound. This closure can be very rapid if vitreous is escaping or prolapsing. The manner in which it is placed approximates more closely than any other the accurate apposition that one expects and obtains in other surgical procedures. It is more perfect mechanically than other sutures in that, with it, the incision is sutured rather than pulled together. If properly placed, there is no danger of overriding or lateral displacement. Secure closure eliminates the disadvantages of a large incision.

2. With such accurate closure, incisions heal much more rapidly and the anterior chamber is reformed much earlier. It is not uncommon to find, after the ten minute interval spoken of, that the anterior chamber has an appreciable depth. Almost invariably it is of normal depth on the first inspection, from forty-eight to seventy-two hours after the operation, and in cases that have required inspection a few hours after operation, the chamber has been found to be of nearly normal depth.

3. The Van Lint flap covers the entire incision, tending to prevent exogenous infection. Iris prolapse, which cannot be absolutely prevented by any single suture, is already covered by a conjunctival flap. In the fifty or more cases in which this type of closure has been used, there have been only two cases of iris prolapse, one in an extremely obstreperous patient, the other following the shock of information of the death of the patient's husband.

In a personal communication, Dr. F. H. Verhoeff pointed out that the Van Lint flap as we use it lessens the danger of growth of the conjunctival epithelium into the anterior chamber, since there are no small flaps of conjunctiva left at the limbus to turn inward when the incision is closed.

4. By using Derby's scleroconjunctival suture in conjunction with the Van Lint flap, the difficulty of obtaining a safe closure after preliminary iridectomy is eliminated.

The disadvantages of this method are far outweighed by its advantages. The chief disadvantages are: 1. The Verhoeff suture is difficult to place properly. It is true that this stitch is a little more difficult to place than some of the others, but the difficulty soon disappears with a little practice. Objections to efficacious surgical procedures on the grounds of technical difficulty are not valid.

2. The time of operation is lengthened. This is the main disadvantage of the procedure, but to offset it one has the assurance that the incision is properly closed and adequately protected. In Lancaster's [9] discussion of modern methods of ocular anesthesia, he said: "No longer is speed an all important consideration . . . instead, we can take time . . . confident that if anything calamitous occurs, it will not be the fault of the patient but of the operator or his assistant. . . . Hence the confidence with which we attack complicated cases, doing operations which under the old conditions would have been not only bold, but even reckless."

3. The free dissection of the conjunctiva undoubtedly increases the postoperative reaction somewhat, but not seriously.

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9. Lancaster, W.: Surg., Gynec. & Obst. **55**:452 (Feb.) 1931.

Other objections to the Verhoeff suture have been raised, viz., that astigmatism is increased, that severe reactions are likely to occur in the cornea and that the suture may serve as an avenue of infection.

In our experience, astigmatism has been lessened rather than increased. This is due, we believe, to the fact that the suture prevents any lateral displacement or distortion of the section and prevents bulging of the incision. No severe corneal reactions have been seen, either here or in the junior author's service under Verhoeff, covering a total of perhaps three hundred cases. Frequently a fan of fine corneal striae, radiating from the point of exit of the suture, appears, but the striae invariably clear up in a few days. No infections traceable to the suture have been seen, nor does it seem likely that they could occur since the suture does not penetrate the cornea and is at all times covered by the conjunctival flap.

The removal of the suture has been cited as a disadvantage of its use. This point has been discussed by both Ellett and Howard. To quote Ellett,[6] "The patient need not know there is a stitch in the eye, at least until the time comes for its removal, and then the information, casually imparted as a matter of course, will not strike him as anything out of the way, since sutures are usually put into wounds." We are in entire agreement. Our patients, we find, accept the presence of sutures as a matter of course, and, in fact, we frequently use their presence as a convenient peg on which to hang the unavoidable post-operative irritation, to the benefit of the patient's peace of mind.

SUMMARY

1. The various types of sutures are briefly discussed.

2. Various indications that must be met in closing the large incision required in intracapsular extraction are discussed.

3. The application of a combination of the Verhoeff corneal-sclero-conjunctival suture with the Van Lint sliding flap is described.

4. The combination of Derby's modification of the original Verhoeff scleroconjunctival suture with the Van Lint flap permits a secure closure of the cataract incision following a preliminary iridectomy.

TULAREMIA

SOME PATHOLOGIC AND CLINICAL MANIFESTATIONS, WITH A REPORT OF TWO CASES

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This report of tularemia occurring in two brothers, with ocular lesions in one, is made because of the comparative infrequency of the reports of ocular lesions in this disease, and also because of the occurrence, in both cases, of some rather unusual and interesting allergic manifestations.

No details of the history and etiology of tularemia are repeated here, as they have been fully described in the literature.

REPORT OF CASES

CASE 1.—L. C., a colored boy of 17 years, a high school student, presented himself at the eye clinic of Rush Medical College on Dec. 12, 1931, because of pain and redness of the left eye, with swelling of the lids, and a tender swelling in front of the left ear of about two days' duration. The patient attributed the ocular inflammation to an injury to the left eye which occurred two days earlier, when it was accidentally struck by a flying piece of wood during a manual training class at school. On further questioning as to any contact with rabbits or other animals (suggested by the finding of ulcers of the conjunctiva), the patient recalled that eight days earlier, on December 4, he had assisted his brother, an employee of a butcher shop, in skinning and dressing three hundred wild rabbits.

Physical Findings.—The general appearance and facial expression of the patient suggested acute illness, and the oral temperature was 100.8 F. On the left side, the preauricular, the parotid and the superior cervical (lymph) glands were markedly enlarged, hard and tender.

Ocular Findings.—Visual acuity was: O. D., 0.8; O. S., 0.6. The right eye was normal. The left eye presented acute pathologic changes of the eyelids and conjunctiva as follows: There were marked swelling and redness of the lids, and marked injection of the palpebral and bulbar conjunctiva. There were numerous sharply defined yellowish nodules, from 2 to 3 mm. in diameter, in both the upper and lower tarsal and transitional fold conjunctivae. The outer thirds of the upper and lower palpebral conjunctiva each presented a 3 by 5 mm. ulcerated area, the floor of which was coated with yellow, necrotic material. There were two yellowish nodules in the bulbar conjunctiva 3 and 4 mm. respectively from the temporal limbus, and another one nasally near the semilunar fold (fig. *A*). There was no secretion in the conjunctival sac. The cornea, iris, media and fundus were normal.

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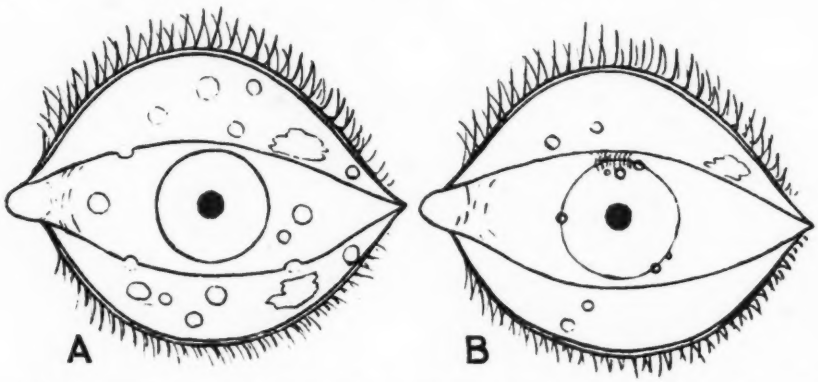
General examination revealed (in addition to the acutely enlarged glands noted) a left acute suppurative otitis media and a marked acute tonsillitis and pharyngitis. Scattered over the free surface of each tonsil were irregularly outlined, yellowish patches, covered by a thin, grayish membrane. The exudate discharging from the left ear was sanguinopurulent. The tympanic membrane was found already perforated. The leukocytes of the blood were increased to 11,000. There were no pathologic internal findings. The patient was admitted to the Presbyterian Hospital on December 13.

Clinical Course.—Four weeks after the onset of the disease, suppuration of the enlarged glands began, and on incision thick white pus was obtained. Until the end of the second week there was remittent fever as high as 103.8 F. At the end of the third week the temperature had reached a normal level, while during the fourth week it gradually rose again to evening temperatures of 101 F., with morning remissions down to normal. This type of fever remained for eight weeks, after which time the temperature gradually returned to normal. During the fourth week three limbal, pinhead-sized nodules at 4, 5 and 9 o'clock developed, and two nodules appeared in the cornea of the affected left eye, 2 and 3 mm. from the



A, left eye of L. C. (case 1) four days after the onset of the disease. *B*, appearance four weeks after onset.

upper limbus, all of them accompanied by a sector-shaped injection of the corresponding bulbar conjunctiva. In the fifth week another limbal nodule appeared at 1 o'clock, and superficial vessels had invaded the cornea with superficial infiltration forming a pannus between the upper limbus and the corneal nodules (fig. *B*). By this time only a shallow loss of substance remained at the former site of the conjunctival ulcers. Also most of the conjunctival nodules had disappeared without ulceration. The corneal and limbal nodules disappeared within two weeks, leaving for some time, however, small, nonelevated grayish opacities at their former site.

Case 2.—W. C., aged 18, a grocery clerk and a brother of L. C., our first patient, presented himself at the eye clinic of Rush Medical College on Dec. 31, 1931. About three weeks before, the patient had dressed wild rabbits several days in succession. In some of these animals the abdominal cavity, liver, spleen and peritoneum were studded with white spots. Two weeks before examination a feeling of general malaise with a head cold and an inflammation of the left eye developed. There was some mucoid discharge from the eye, which subsided after two days. At the same time a swelling on the tip of the right middle finger, accompanied by a sharp pain, and a swelling in the left axilla had developed.

Physical Findings.—The patient appeared weak and apathetic, and the oral temperature was 101.8 F. The tip of the right middle finger was moderately swollen, and an ulceration occupied one side of the tip, also undermining the nail. The medial surface of the forearm as well as the upper part of the arm was tender, and in the right axilla there were two or three 3 by 4 cm. hard and tender glands. No other glands were enlarged. There were no ocular changes. A leukocytosis of 12,000 was found. An indefinite, deep tenderness along the left costal margin was present, but further internal examination did not reveal any pathologic conditions. The patient was admitted to the Presbyterian Hospital on the day of examination.

Clinical Course.—During the following week the temperature was of a remittent type, as high as 103 F. The axillary glands suppurated and were incised during the fourth week of the illness. Five weeks after the onset of the disease the temperature was normal again, the ulcer on the finger was healed and the patient was discharged, with only slight remaining discomfort in the right axilla. During the fifth week small tender nodules, three or four at a time, had appeared on various parts of the body, mostly on the left side, according to the patient. They lasted one or two days and then disappeared completely, while others arose at a different place. One was pointed out by the patient on the back of the right wrist. This was a firm, insensitive swelling measuring 1 by 1 cm., which had been tender the day before. The skin was freely movable over it and did not show any redness. No other eruption could be detected at any time during the illness. The aforementioned nodule on the back of the wrist disappeared after five days.

Bacteriologic and Serologic Findings.—The serologic diagnosis of tularemia in case 1 was first made in the Illinois State Laboratories on December 23, sixteen days after the onset of the disease. The titer of the agglutinins found in the serum at that time was 1 : 320. On January 3, eleven days later, the serum tested in the hospital laboratories had a titer of 1 : 3,560. Serum from the patient in case 2 reached a titer of 1 : 640 thirty-six days after the onset of symptoms. There was a moderate agglutination of A. melitensis and A. abortus antigens in a dilution of 1 : 40 in serum from the patient in case 1.

Pus from the finger of the second patient was taken on January 5, three weeks after infection. Cultures were made, and a guinea-pig was inoculated subcutaneously. The only organism recovered on blood agar was Staphylococcus albus, but the animal died in seven days. Gross examination showed hemorrhagic subcutaneous tissues; the spleen and liver were enlarged and firm and studded with pinpoint-sized, slightly raised, yellowish nodules such as have been found to be characteristic of tularemic infection in man and animal (liver, spleen, meninges [1]). There was a serous pericarditis. There were no enlarged glands and no pneumonia. In smears of the spleen there were many minute gram-negative organisms which were recovered from both the spleen and heart's blood in pure culture on cystine dextrose blood agar. This organism does not grow on a plain agar. It was agglutinated by the serum from the patients in cases 1 and 2 in dilutions of 1 : 1,280 and 1 : 320, respectively, and killed a guinea-pig inoculated on the scarified surface of the abdomen in six days with the lesions described for the first animal and the additional involvement of the inguinal glands and lungs.

Pus was taken from the involved glands of both patients on January 13, and inoculated subcutaneously in guinea-pigs. These animals are living and well after

1. Bryant, A. A., and Hirsch, E. F.: Tularemic Leptomeningitis, Arch. Path. **12**:917 (Dec.) 1931.

twelve weeks. Parts of the spleen and liver of the first guinea-pig were embedded in celloidin and sectioned; the sections were stained with hematoxylin-eosin.

The malpighian bodies of the spleen exhibited the most severe changes. The lymphocytes in the germ centers had lost their shape and staining properties, and heaps of cell débris indicated a rapid cell disintegration; multiple, pinhead-sized areas of simple necrosis were scattered throughout the pulp, about seven or eight to one low power field. There was no caseation necrosis or giant cell formation. Surrounding many but not all of these focal necroses was a narrow zone of fibrocytes and fibroblasts.

The changes in the liver were less pronounced. Minute infiltrates could be detected along the intralobular and interlobular blood vessels and some of the biliary ducts, and also between the cords of hepatic cells, which were completely necrotic in these places. The smallest infiltrates had a purely mononuclear character, while in the large foci a few polymorphonuclear cells were also present. Smears made from the conjunctival ulcers and also from the content of two nonulcerated nodules of the palpebral conjunctiva were stained by the Gram-Weigert method and found negative for Bacterium tularense.

COMMENT

In only three of the thirty-six reported cases of the oculoglandular type of tularemia were there lesions of the bulbar conjunctiva. In Clark's [2] case a single ulcer in the bulbar conjunctiva, from 4 to 5 mm. in diameter, was the only ocular lesion; Vail [3] reported one case of a limbal nodule encroaching on the cornea for 2 mm. Sattler [4] made a histologic examination of one nodule, which he had found in the bulbar conjunctiva during the fifth week of the illness, after all the other ocular and general symptoms had subsided. The excision of this nodule brought about an immediate recrudescence of the same local and constitutional symptoms with which the patient had first been seen. Half of this nodule was used for the inoculation of a guinea-pig, which remained healthy.

Corneal lesions have been reported in three instances. In Francis' [5] case a corneal ulcer led to perforation and prolapse of the iris. Pfunder [6] described a steamy cornea in one of his patients, without giving further details. Judd [7] saw a dendritic keratitis, accompanied by a mild iritis, develop during the first week of illness. Within two weeks the keratitis healed under the usual treatment of heat, atropine and corrosive mercuric chloride ointment.

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2. Clark, C. P.: Atypical Conjunctivitis Tularensis, Am. J. Ophth. **11**:280, 1928.

3. Vail, T. D., Jr.: B. Tularense (Squirrel Plague) Conjunctivitis, Arch. Ophth. **55**:235 (May) 1926.

4. Sattler: Bacterium Tularense Conjunctivitis, Arch. Ophth. **44**:265, 1915.

5. Francis, E.: Tularemia, in Tice: Practice of Medicine, Hagerstown, Md., W. F. Prior Company, Inc., 1928, vol. 3, p. 663.

6. Pfunder, M. C.: Primary Tularemia of the Eye, J. A. M. A **85**:1061 (Oct. 3) 1925.

7. Judd, J. H.: Ocular Lesions in Tularemia, Arch. Ophth. **2**:300 (Sept.) 1929.

There seems to be a certain parallelism in the development of the nodular efflorescences in our two cases. They appeared as late as the fourth and fifth week after the onset of the disease, and were of a transitory character. In case 1 they were similar to the phlyctenules of eczematous keratoconjunctivitis, or to those of repeated experimental instillations of horse serum into the conjunctival sac, resulting in anaphylactic manifestations.

Subcutaneous nodular lesions simulating those of sporotrichosis, which follow the lymphatic vessels from the primary lesion to the regional glands, have been described by Hodge,[8] and Freedlander and Grossberg.[9] These nodules are at first deep and indurated, but may later suppurate as do the nodules of sporotrichosis. While nodules on cornea and limbus appearing during the later stages of the illness have been described in a few instances, no record was found of a subcutaneous lesion similar to the evanescent nodules in our case 2, which have much in common with the efflorescences of erythema nodosum, also an allergic manifestation. The striking clinical similarity of the ocular nodules to scrofulous phlyctenules and anaphylactic keratoconjunctivitis suggests the idea that also in tularemia there may be a basic sensitization such as in tuberculous allergy or after injections of an antigenic substance such as horse serum. A parallel condition may possibly arise in the ulceroglandular or pure glandular type with respective manifestations.

One could assume that by the fourth week the tissues are immunologically sensitized by the tularemia infection, and that the action on these tissues of certain toxic substances present in blood, lymph and perhaps lacrimal fluid (Riehm[10]) brings about an eruption similar to phlyctenules or erythema nodosum.

SUMMARY

1. One case of the oculoglandular type of tularemia with corneal involvement is added to the three hitherto reported cases with corneal lesions.

2. From the focal lesion in an ulceroglandular case cultures were isolated through a guinea-pig at the end of the third week.

3. The eruption of nodular efflorescences during the fourth and fifth week of the illness in both cases is compared with similar manifestations in allergic conditions.

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8. Hodge, F. C.: Three Cases of Tularemia, One Resembling Sporotrichosis, Am. J. M. Sc. **170**:57, 1925.

9. Freedlander, S. O., and Grossberg, M. H.: Tularemia, J. A. M. A. **88**:475 (Feb. 12) 1927.

10. Riehm, W.: Ueber experimentelle anaphylactische Keratoconjunctivitis am Kaninchenauge, Arch. f. Augenh. **99**:438, 1928.

FOREIGN PROTEINS AND GOLD IN THE TREATMENT OF UVEITIS

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Chronic inflammation of the uveal tract usually is resistant to treatment and is characterized by periods of quiescence after an exacerbation of varying degrees of activity. The causes most frequently ascribed are focal infection, syphilis and tuberculosis. Other causes are injuries, foreign bodies, local diseases of the eye, such as corneal ulcers, abscess of the lids, tumors of the orbit and cellulitis. Focal infection may be the activating agent in association with one or more of any of the other probable causes, and in many cases its presence tends to confuse the part played by any other factor. In the absence of clinical and serologic evidence of other systemic disease, the manifest presence of infection about the teeth, tonsils and pelvic organs is sufficient to ascribe the current attack to such infection. The prompt improvement in the eye following the eradication of all foci of infection that can be removed strengthens the assumption that the disease of the eye was metastatic.

The presence of clinical or serologic evidence of syphilis in cases of uveitis cannot be ignored. The newer knowledge of the clinical aspects of secondary and tertiary syphilitic lesions as compared to the lesion of focal infection in cases in which syphilis is not present leads to the conclusion that the disease of the eye may be the result of the combined action of syphilis and the infection. Treatment of either condition may bring about a satisfactory response in the eye, thus clouding the etiology with the doubtful part of the two possible causative factors.

The finding of tuberculosis in any form has not the clinical significance of the finding of a positive serologic reaction for syphilis. Except for the finding of miliary tubercles in the choroid and iris, tuberculosis of the eye is proved in comparatively few cases. Many diseases of the eye formerly attributed to tuberculosis by writers on clinical ophthalmology are now known to belong to some other category, or at least the incidence of those diseases definitely shown to be tuberculosis is gradually decreasing.

It is generally conceded by clinicians that tuberculosis and focal infection may escape detection, and one can seldom be sure of the absence of either condition. A small focus may produce symptoms that

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recur again and again in a highly susceptible person. The low grade inflammations of the uvea that become chronic are of this type.

The milder forms of diseases of the uveal tract are likely to be still more confusing. There are types of lesions of the choroid that occur in association with syphilis and tuberculosis with such regularity as to be characteristic. The situation, course and duration of the lesions define their character. They may be classified on their appearance by ophthalmoscopic examination. The presence of many infections does not materially alter them. A much larger group, however, is indeterminate. General examination may not reveal adequate evidence of systemic disease or of focal infection, yet the lesions in the choroid may multiply, the iris may become more atrophic, the ocular fluid may become more cloudy, and the lens may undergo cataractous changes. These milder forms of uveitis are found more frequently among persons in the fourth and fifth decades of life. Young persons are less frequently affected than the aged. A difference in incidence has not been noted in the sexes.

In any disease of the iris and ciliary body the choroid is more or less involved, as the blood current is from the anterior to the posterior part of the uvea. Isolated lesions of the choroid, however, may be quite extensive without producing noticeable changes in the iris. A wide variety of pathologic changes are designated by terms that indicate their situation and character, yet the essential features of the disease that produces these changes are common to almost all of them. Without an attempt to enumerate or classify inflammations of the iris, ciliary body and choroid, they may all be grouped as diseases of the uveal tract and from the standpoint of treatment may be in the same category.

Except for conditions that have a definite and obvious cause, the treatment of diseases of the uveal tract is often necessarily prolonged and hence difficult to carry out long enough to secure the best results. Specific treatment rarely is possible. Removal of focal infection, if possible, or treatment of infected organs is indicated. Antisyphilitic treatment may be sufficient if the patient has a positive serologic reaction, but the treatment of uveal tuberculosis or mixed types of uveitis requires patient and persistent medication. Tuberculin in various forms is widely used and justly so if unfavorable reactions do not occur. However, the frequency with which unfavorable reactions do occur prohibits its use in many cases for fear of exciting dormant tubercles to activity, with resultant spread of local injury.

The form of treatment that has been employed in the Mayo Clinic for diseases of the uveal tract has been worked out on the therapeutic response to nonspecific protein or metallic substances selected for use in a given case on the basis of activity of the lesion and the reaction produced by the drug. Antisyphilitic treatment is used if serologic tests

are positive. A few cases of uveitis of local origin, such as corneal disease, may be treated only by local measures, but the majority of cases are recurrent or chronic, and often of indeterminate origin, and hence must be reached through systemic treatment. As a rule, the more acute forms of uveitis demand more vigorous treatment. The most effective form of treatment we have today of combating localized acute infection is foreign protein. Many substances have been made available for parenteral injection from which varying degrees of thermal reaction may be expected, and antibodies of nonspecific character may be generated or exfoliated. It has been stated that a thermal reaction is not necessary to obtain a satisfactory therapeutic effect, and some substances have been recommended because of the slight fever resulting from their administration. This is, however, a virtue of questionable value. Milk may be taken as a standard from which to judge the effect of other protein substances. The rise in temperature following the intramuscular injection of 10 cc. of boiled whole milk varies from less than 1 degree to 6 degrees. In a series of acute diseases of the eye treated at the clinic, the therapeutic effect was not comparable to the rise in temperature.[1] In some cases relief of pain followed shortly after the first injection, and diminution of inflammation was noticeable within a few hours; in none, however, was there any evidence that a rise in temperature hindered or delayed healing. We have come to look on chills and fever, after the injection of milk, as inconsequential. Of all the substances used for quick results we have come to the conclusion that whole milk is the most reliable and the safest.

Boiled milk is injected in amounts from 6 to 10 cc. into the gluteal muscles in cases of acute anterior uveitis of short duration. A second injection is not given until twenty-four hours after subsidence of any rise in temperature that may have been induced. The maximal effect is obtained after four or five injections. No advantage has been gained by giving more than 10 cc. at a time. If the eye has not become free from inflammation by the end of the reaction following the fifth injection, the disease becomes subacute and falls into the second group.

There is no sharp line between the three grades of activity or duration of inflammation that indicates the line of treatment to be used. The second or subacute group is comprised of cases of low grade uveitis in which the condition has been active for one month and not more than three months. This does not refer to recurrent attacks. As this group is rather elastic, cases of acute inflammation will be included in which, in spite of vigorous treatment, the condition continued into

1. Benedict, W. L., and Rucker, C. W.: The Use of Foreign Proteins in the Treatment of Diseases of the Eye, New Orleans M. & S. J. **81**:782 (May) 1929.

the second month, and likewise mild cases of several weeks' or months' duration with but little change of tissue and comparatively small loss of function. We also include in this group lesions of the choroid of recent origin. The effect of milk on this group has not been encouraging. It is necessary to extend treatment longer than the time milk seems to be effective. Consequently, we employ typhoid vaccine intravenously, or autogenous or stock vaccines subcutaneously. The choice of vaccine depends largely on the desired intensity of the reaction. Typhoid vaccine almost always produces chill and fever after the first few injections. The fever may run dangerously high, more so than with milk given intramuscularly; therefore, it must be considered less safe. However, with properly regulated dosage, any degree of reaction desired can usually be approximated. Injections of typhoid vaccine may be continued at a constant level of doses of from 25,000,000 to 50,000,000, or the dosage may be stepped up rapidly to 500,000,000. The reasons for changing dosage are rather arbitrary, but it is believed that a rapid increase in the strength of the dose produces more violent reactions and induces more rapid healing. The use of fever-producing proteins is a safe procedure in the treatment of inflammations of the uveal tract from any cause in the acute and subacute stages of the disease. The marked reactions following their use cannot, however, be tolerated for more than a few weeks at the most, so that some other form of treatment must be substituted for the chronic form of the disease. Autogenous and stock vaccines are used following the removal of foci of infection and after discontinuing injections of milk or typhoid vaccine for a patient whose eye is slow in becoming clear, but they are not always tolerated even in small doses, and care must be exercised in their use. The unsatisfactory effect of injections of foreign protein in chronic uveitis led me to search for a more suitable substance that could be used for a longer time, and one that was free from the reaction that produced fever.

In cases of chronic uveitis of more than three months' duration the media may be very cloudy and the iris atrophied. Tuberculous uveitis usually is of this type. The disease runs a long course, often with moderate discomfort and loss of vision. Tuberculin is used with good results in some cases, but is not effective or is not tolerated in others. Many physicians hesitate to use tuberculin therapeutically if an active lesion thought to be of tuberculous origin can be seen in the eye. The fear of lighting up a quiescent focus of old tuberculosis by tuberculin is well founded. Its employment in cases of choroiditis is therefore limited or contraindicated. It is also well known that some persons with tuberculous lesions in the uveal tract do not improve following the use of tuberculin, but on the contrary the disease remains fairly stationary

or slowly progressive. Such cases we have placed in a third group, for which treatment should be extended over a period of several months. In this group also are all other cases that have not become quiescent after the use of foreign protein and local treatment of the eye. The eye usually presents mild evidences of inflammation of the iris and ciliary body, such as circumcorneal injection, keratitic precipitates, congestion or atrophy of the iris and opacities of the vitreous. If the lesion is posterior, external signs of inflammation and pain may be absent. Through the cooperation of the Section on Dermatology and Syphilology we have been giving intravenous injections of sodium gold thiosulphate to patients in this group.

Heavy metals have long been in use for their nonspecific as well as their possible specific effect. Interest in treatment by gold preparations was revived when Möllgaard recommended sodium auro-thiosulphate for pulmonary tuberculosis. Its lack of efficacy in this form of tuberculosis is now universally conceded, but it has shown itself of real merit in the tuberculids and particularly in lupus erythematosus. Although the latter disease has not been proved to be of tuberculous origin, there is much evidence that tuberculosis is one factor, at least in many cases. The other factor usually considered is either Streptococcus or Staphylococcus. Whatever the exact etiologic agent in lupus erythematosus, it is generally conceded that the pathologic process involves primarily the vascular system of the entire cutis. So severe is this at times that considerable hemorrhage may be found. In the main the changes are chronic, suggestive of a granuloma. In this disease the results from injections of a gold compound have been satisfactory, at times phenomenal. Since many chronic inflammations of the uveal tract are undoubtedly transmitted by way of the vascular system and chiefly involve the vascular system of the eye, injections of a gold preparation were thought worthy of a trial.

The action of gold is not as yet understood, and therefore its therapeutic mechanism is not explainable. At best the response to its use is slow; it must, therefore, be given for a considerable time. Often, if well tolerated, the patient will experience improvement in his general health. It probably does not act directly on an infecting microbe, but rather in some manner stimulates the defense mechanism of the body. Its action is therefore not specific, but rather nonspecific. Gold is a capillary poison, and, as such, its therapeutic use may be explainable, but it has a paralyzing effect on the contractile elements of the capillaries, thus producing peripheral hyperemia. Gold preparations of various types are on the market. In this country, at least in the last few years, sodium gold thiosulphate has chiefly been employed, and in our treatment of diseases of the uveal tract this combination has been used exclusively.

The method of administration is simple. The drug is given intravenously in doses of from 10 to 100 mg. from two to three times a week. The average dose is from 25 to 50 mg. A hundred or more injections may be given. In our experience less than twenty-five injections has not proved of much value. At intervals of several months a course can be repeated. In the smaller doses the drug is not very toxic. It should be remembered, however, that a heavy metal is being administered to patients of varying degree of sensitivity. Unexpectedly a certain patient may prove to be markedly hypersensitive. Every patient treated should be carefully watched, and on the first sign of hypersensitiveness the drug should be stopped at once. The untoward early symptoms and signs and the later complications are those of intoxication by the heavy metals, and can be compared to those seen in the administration of the arsphenamines.

REPORT OF CASES

CASE 1.—A girl, aged 19 years, registered at the Mayo Clinic on April 9, 1928. She had had good vision in both eyes until six years previously; since then the vision in the left eye had gone entirely and had been failing in the right eye since the previous winter. Vision in the right eye was 6/20; in the left eye, nil. A diagnosis was made of posterior uveitis in both eyes, and cyclic cataract. A roentgenogram revealed evidence of pulmonary tuberculosis. From April 13 to July 25, 1928, the patient was given twenty-six injections of a gold preparation, two of 25 mg. and twenty-four of 50 mg. By April vision in the right eye had improved to 6/12—1, but the left eye was the same; on July 10 vision in the right eye was 6/10, and that in the left eye was nil. The patient was dismissed on July 25, with vision in the right eye 6/7—2 and that in the left eye nil. There was no further report from this patient.

CASE 2.—A physician, aged 28, registered at the clinic on May 5, 1928. He gave a history of vitreous hemorrhages in the left eye three months previously which cleared up in one month and recurred one month later. An area of subacute choroiditis in the lower quadrant of the nose was present. The diagnosis was choked disks, with vitreous hemorrhages in the left eye. General examination revealed evidence of old tuberculosis with no present activity. The patient was given a course of four injections of milk, of 10 cc. each. Vision in the right eye was 6/6, and in the left eye, 6/60. From May 15 to July 25, 1928, the patient was given twenty-one injections of gold, four of 25 mg. and seventeen of 50 mg., and was dismissed from observation. In August, he wrote to say that there were no further hemorrhages and that the opacities were absorbing. In December, 1930, his eyes were refracted and a few fine vitreous opacities were noted.

CASE 3.—A woman, aged 29, registered at the clinic on April 22, 1929, with a complaint of inflammation of both eyes of six weeks' duration; the left eye was affected first, and the right eye, two weeks later. The patient experienced some photophobia but no pain. The diagnosis was iritis of both eyes. Vision in the right eye was 6/12; in the left eye, 6/30. The general examination yielded negative results. The patient returned to the clinic on July 8 with vision failing. Vision in the right eye was 6/15; in the left eye, 6/20. Heavy, stringy, dustlike opacities were found in the vitreous. The patient was given a course of eight injections of 25 mg. each of a gold preparation. One month later there was no change in

vision; the gold preparation was discontinued and potassium iodide was given. On September 23, vision in the right eye was 6/15; in the left eye, 6/10. The vitreous opacities were about the same. Iritis had not recurred. Treatment with potassium iodide was continued. On November 18, the opacities were much clearer in the left eye. Vision in the right eye was 6/6—1; in the left eye, 6/15—1. On December 2, a few fine vitreous opacities were observed in both eyes. Marked improvement had occurred in the last thirty days. Potassium iodide was discontinued, and treatment with ethylmorphine hydrochloride was instituted. On June 2, 1930, vision in both eyes was 6/6 without correction; some fine floating opacities were still present. No further report was received from the patient.

Case 4.—A woman, aged 34, registered at the clinic on Oct. 5, 1927. She was able to count fingers at a distance of 2 feet with the right eye; vision in the left eye was 6/60. The diagnosis was active choroiditis in both eyes. No focus of infection was found on general examination; cultures from the cervix showed Streptococcus, Staphylococcus and gram-positive bacilli. Treatment with sodium salicylate, diphtheria antitoxin and arsphenamine was instituted. Three months later, after six treatments with arsphenamine, vision in the right eye was 4/60. Six months later the patient returned with central vision low and peripheral vision good. Further treatment with arsphenamine was given. Two months later the exudate in the fundus was gradually receding and treatment was discontinued. The patient returned on Nov. 5, 1928, with vision unchanged. From then to Feb. 13, 1929, she was given forty-two injections of a gold preparation, six of 25 mg. and thirty-six of 50 mg. Central vision did not improve, the scotomas were smaller, and the patient was dismissed without further treatment. Eight weeks after the last treatment with the gold preparation there was not much change in the appearance of the fundus of each eye. On Sept. 17, 1929, the patient was given two injections of gold, 25 mg. each, and was to continue treatment at home. On May 12, 1930, sixty doses of gold had been taken. Seven months later the patient renewed the injections of the gold compound, taking sixty more. On June 17, 1931, vision in the right eye was 3/60; in the left, 6/30. Treatment was discontinued. On December 17, vision in the right eye was 1/60, and in the left, 6/30; there were central scars in the choroid, and the disease was arrested.

Case 5.—A man, aged 34, registered at the clinic on Feb. 7, 1929, with the complaint of sore eyes for the last five years after coal dust had blown into one eye, producing corneal ulcers. The eyes were painful at intervals. There was a gradual diminution in vision, which was more rapid during the last year. Vision in the right eye was 3/60; with the left eye, moving objects could be seen. The diagnosis was marginal keratoconjunctivitis of the right eye. The patient gave a history of an old tuberculous gland. The Mantoux test for tuberculosis was positive. From February 15 to 27, he was given four injections of sodium gold thiosulphate with much resultant improvement. On February 28 the eye was very uncomfortable and the injection of gold was stopped because of intolerance. The condition of the eye continued to flare up until April 11, when the patient was dismissed. Vision in the right eye was 6/60; with the left eye, moving objects could be seen. The patient was given atropine, mercurochrome-220 soluble and ethylmorphine hydrochloride. On July 11, 1930, he wrote that the right eye was much better and that he could see to read and was able to work every day. On Oct. 22, 1931, he wrote again to state that the right eye was clear and gave him no trouble.

Case 6.—A man, aged 45, registered at the clinic on Dec. 28, 1928, with a complaint of floating opacities in both eyes for the last four years. The onset was insidious and not associated with illness or accident. There were numerous remissions of the condition, but on the whole vision had been getting worse. The patient

complained of severe migraine. Vision in the right eye was 3/60; with the left eye, moving objects could be seen. The diagnosis was mild uveitis with vitreous opacities in both eyes. General examination revealed no systemic disease, and on Jan. 9, 1929, a series of injections of a gold preparation was begun. From that time until February 13, he received fifteen injections of 25 mg. each. Vision improved subjectively, especially in the right eye. The patient was dismissed, to continue the treatment with the gold preparation at home to a total of thirty injections. He returned on April 29 with vision in the left eye much improved. Vision in the right eye was 3/60; in the left eye, 6/6—2. The patient reported by letter that the eyes improved until July 31, 1929, when another hemorrhage occurred. In September he reported monthly recurrences. In November he reported that the eyes had not cleared up as before following a hemorrhage. On Jan. 1, 1930, he wrote that he had had no recurrences since Dec. 1, 1929. In March, 1930, he reported that the eyes were clearing and that he had had no further hemorrhages. In August, 1930, the eyes were still good. In December, 1930, he wrote that he believed the eyes were steadily gaining. He returned to the clinic on Aug. 22, 1931. Vision in the left eye had been good until three weeks before. Presumably another hemorrhage had occurred. The diagnosis was retinitis proliferans in the left eye. General examination revealed septic tonsils, infected teeth and a scar on the neck which was thought to have resulted from old tuberculous adenitis. On December 7, the patient reported that sixty days after leaving the clinic the eyes had cleared so that he could easily read the newspapers. No further report has been received.

ABSTRACT OF DISCUSSION

DR. GEORGE SLOCUM, Ann Arbor, Mich.: The value of paraspecific therapy is well established. In 1919, D. M. Cowie (Nonspecific Therapy in Arthritis and Infections, *Arch. Int. Med.* **23**:69 [Jan.] 1919) published his researches on the use of typhoid vaccine as a nonspecific remedy. In September, 1919, there occurred in our clinic a postoperative cataract infection which was referred to Dr. Cowie for typhoid vaccine. Since that time we have used this remedy in an increasing variety of cases. It has been our experience that the most benefit follows a systemic reaction; in some cases improvement has taken place when no reaction occurred.

In our experience while injections of milk have proved beneficial in many cases, they have not appeared to be superior to typhoid vaccine. We prefer typhoid protein because of its smaller bulk and because it can be obtained in standardized form. Dr. Benedict's experience that the acute forms of uveitis respond better to nonspecific therapy agrees with our own. Also we agree that there is no sharp line of demarcation between cases that will and those that will not respond to this treatment. To avoid marked reaction we give the first dose subcutaneously; if no severe reaction follows, subsequent doses are given intravenously. If considerable reaction follows, the subcutaneous injection is quite as effective, and it is continued. Long ago, Wright stated: "No one recovers from a bacterial disease unless protective substances are produced in his organism." This is the effect that we have in mind in foreign protein therapy.

Our experience with the use of tuberculin in suitable cases has been rather satisfactory. To avoid untoward effects we have given relatively small doses, not too frequently repeated, and have thus avoided the "negative phase" described by Wright in his "Studies on Immunization."

With the use of colloidal preparations of the heavy metals in the treatment of tuberculous or other forms of uveitis, I have had no experience.

According to Udo J. Wile, sodium gold thiosulphate is of no value in tuberculosis or syphilis. On the other hand, in lupus erythematosus, the beneficial effect

is often pronounced. As to why gold salts are beneficial, there is, as Dr. Benedict says, considerable uncertainty. Some investigators believe that gold acts as a nonspecific ferment, stimulating the production of antibodies and body reactions comparable to the effects of nonspecific proteins. Therefore I was much interested in Dr. Benedict's experience with the gold and sodium preparation.

DR. ALAN C. WOODS, Baltimore: I have had no personal experience with the use of sodium gold thiosulphate in ocular disease, but I had the opportunity of following a case in which this drug was given for another reason, and I think the case sufficiently important to be reported at this time.

A small boy with lupus of the skin was admitted to the dermatologic clinic of the Johns Hopkins Hospital. In this clinic intravenous treatment with sodium gold thiosulphate had been used for this condition for some time without complications. The patient was similarly treated. He had a violent reaction, with the occurrence of small papules over mucous membrane surfaces of the body, nose, lips, conjunctivae, corneas and mouth and over the genitalia. These papules soon ruptured and those on the corneas became secondarily infected. Bilateral corneal ulceration developed; this resulted in perforation with subsequent panophthalmitis and ultimate loss of both eyes.

I understand that occasional severe reactions from the use of this drug have been reported, especially from the German clinics. While this drug is apparently of great value as a therapeutic agent, and the percentage of danger slight, nevertheless, I think that we should bear in mind that disastrous complications, such as I have here reported, may occasionally follow its use.

DR. W. L. BENEDICT: I wish to add two or three points. In the first place, there is no definite indication for the administration of gold or sodium thiosulphate; it is a drug of last resort. We use foreign proteins and other remedies as long as they are effective and do not resort to this until we get no improvement from the other remedies.

Dr. Woods' report of a case is timely. The drug is not for indiscriminate use. It should be given only in cooperation with the dermatologist, and our administrations have been given in the dermatologic section.

We do not look on the use of sodium gold thiosulphate as a cure-all. In fact, many cases have showed no improvement. If, however, after a few doses there seems to be some improvement, with no contraindication for its use, it may be continued at the direction of the dermatologist and ophthalmologist.

It may be secured in ampules of 25 mg., and the dosage is from twenty-five to fifty. We have given it two or three times a week until fifty or sixty injections are given; then it is discontinued for one or two months, and then a second course of fifty or sixty injections is given. If during the early administration there are symptoms of paralysis, the drug should be stopped. We do not look for beneficial effects under two or three weeks. If the heart action is known to be slow the effects are not evident for three or four weeks. We have administered this drug to many people and find that in three or four months after the cessation of treatment there is still continued improvement, and if there are relapses they are likely to be farther apart and not so severe.

SURGERY OF THE ORBIT

GÉZA DE TAKÁTS, M.D.
CHICAGO

It shows unusual temerity on the part of a general surgeon to discuss orbital tumors before ophthalmologists. Throughout this country and in most European clinics, the surgery of the orbit is in the hands of the eye surgeon, who justly claims his right to all orbital contents. It so happened, however, that as a member of the surgical department of the University of Budapest I had the opportunity to study and operate on a comparatively large number of patients, as the Eye Clinic of Professor de Grósz transferred all extra-ocular growths to the surgical department. Aided by a close cooperation between ophthalmologist and rhinologist, I was able to report 109 cases in 1922.[1] Since my departure from the surgical clinic, an additional group of cases has been studied and recently summarized by Prof. T. Verbély.[2] His report appeared in the *Proceedings of the Hungarian Surgical Society,* and only scant abstracts are available in the German literature.

Since changes in the conception of some of these tumors have occurred, I have reclassified both my previous and the more recent material. In a brief discussion, I shall limit myself to a few of the more unusual cases and shall dwell rather on such technical procedures as deviate from customary practice.

A tabulation of the total number of cases (246) reveals the following 5 groups (table 1): congenital anomalies (8 cases), injuries (14 cases), vascular lesions (33 cases), infections (76 cases) and neoplasms (94 cases). Obviously, this is an imperfect classification, as the traumatic arteriovenous fistulas really belong to the injuries, and most of the so-called angiomas are congenital anomalies. Nevertheless, the establishment of a separate vascular group seemed justifiable because of the characteristic circulatory disturbances.

This etiologic classification requires subheadings, which are helpful from the standpoint of diagnosis and therapy. The distinction between intra-orbital, orbitoparietal and extra-orbital lesions is of more than academic interest. Thus in an extra-orbital infection, such as ethmoidal sinusitis, an orbitotomy is of no avail, no matter how pronounced the

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Presented before the Chicago Ophthalmological Society, Jan. 18, 1932.

1. de Takáts, Géza: Causes and Surgical Treatment of Exophthalmus, Post-Grad. M. J. **12**:125, 1922.

2. Verebély, T.: The Surgery of the Orbit, Proc. Hung. Surg. Soc. **16**:1, 1930.

exophthalmos and the retrobulbar edema may be. The eradication of the ethmoidal cells is the logical procedure.

In the group of congenital anomalies, the cerebral herniations or encephaloceles are particularly interesting. Two of them were fronto-ethmoidal, but one was of the posterior variety, coming through the superior orbital fissure. This latter case required a resection of the lateral wall. On pressure on the growth, signs of increased intracranial pressure could be elicited.

Dermoids around the orbital ridge are quite frequent, and I have not included them in the group of 4 retrobulbar dermoids. In spite of a perfectly benign histologic structure, these often multilocular cysts have a remarkably expansive character, enlarging, invading and even

TABLE 1.—*Surgery of the Orbit;* Material of the First Surgical Clinic, University of Budapest, 1914-1930*

Congenital Anomalies	
Cystic microphthalmia	1
Encephalocele	3
Retrobulbar dermoid	4
	8
Injuries	
Foreign bodies	7
Traumatic blood cysts	2
Subperiosteal bone cysts	3
Posthemorrhagic fibrosis	2
	14
Vascular Lesions	
Pulsating exophthalmos	8
Intermittent exophthalmos	4
Angioma	21
(Telangiectasis, cavernous and racemose angioma)	
	33
Infections	
Acute pyogenic	
Cellulitis	12
Osteoperiostitis	46
(Nasal, 40; dental, 6)	
Tuberculosis	9
Syphilis	3
Actinomycosis	1
Echinococcus	2
Chronic inflammatory sclerosis	3
	76
Neoplasms	
Benign	17
Malignant	
Epithelial	19
Mesenchymal	58
	94

* First 109 cases published by me (Post-Graduate **12**: 125, 1922). The whole material was summarized by Verebely (Proc. Hung. Surg. Soc. **16**: 1, 1930).

perforating the bony wall, invading the cranial cavity or the temporal bone. Professor Verbély gave a vivid description of this variety.[3]

There is nothing of unusual interest in the group of foreign bodies (2 bullets, 2 pencils and 3 splinters of wood). One woman, however, blew a bullet out through her nose five days after a suicidal attempt. Exophthalmos is caused by a hematoma, an abscess or an emphysema. Interesting are 3 cases of subperiosteal bone cysts. These cysts contained a viscous bright yellow liquid with cholesterol crystals. A history of trauma on the supra-orbital margin and a fine crack on the superior wall of the orbit were characteristic. These cysts have a tendency to grow. Histologically, the lining of such a cyst consists of cholesterol

3. Verebély, T.: Beiträge zur Pathologie der Augenhöhle, Wien. med. Wchnschr. **79**:1111 (Aug. 24) 1929.

crystals surrounded by foreign body giant cells. Two cases of post-hemorrhagic fibrosis of the orbital contents caused an enophthalmos and simulated a fibrous tumor.

In the third group I have assembled the vascular lesions. Eight cases of pulsating exophthalmos are recorded, 6 of which were traumatic, and 2 probably had an arteriosclerotic basis. Communication between the internal carotid and the cavernous sinus is usually the cause, but occasionally the ruptured aneurysmal sack of the internal carotid may transmit its pulsation to the cavernous sinus, without an actual communication. Cerebral hernias may also transmit their pulsation to the eyeball, but this pulsation is feeble and is influenced by respiration. An aneurysm of the internal maxillary artery filled out the entire maxillary sinus and invaded the orbit through a fissure of the inferior wall, causing pulsation of the eyeball.

The 4 cases of intermittent exophthalmos, i. e., protrusion on bending the head forward, accentuated by jugular compression, were all due to obstruction of venous return in the orbit. In 3 cases varicosities were found, and in 1 case, a small encapsulated angiectasis, which interfered with venous return. Because of the insufficient venous return through the ophthalmic vein, the blood uses the facial-jugular drainage, but when that is obstructed, the increasing protrusion can be easily measured by Hertel's exophthalmometer.

In the large group of angiomas, 21 cases are listed. They are usually classified in the literature as capillary, cavernous and racemose angiomas. My own experience with vascular growths on the extremities has made me feel that these are all congenital anomalies differing only according to the stage in which the arrest of development occurred. Their standstill or sudden expansive growth will depend on the connection with the general circulation, which may be suddenly opened up by trauma, curiously enough, during puberty. Recently their extent has been visualized by opaque substances.

In the large group of infections, it is hardly possible to differentiate between phlegmon, abscess and thrombophlebitis of the orbit in a given case. Aside from accidental and surgical infections, the nasal and dental origin predominates. Interesting is the case of a 16 year old boy in whom periostitis developed around the upper first molar tooth. The periostitis crept up to the inferior wall of the orbit and produced a subperiosteal abscess, causing an exophthalmos of 2 mm. It was easily drained. Of the 18 chronic infections, tuberculosis originated in the bone 14 times and once in the lacrimal gland; syphilis was found as a periosteal gumma twice, and once as a gumma in the external rectus muscle. The case of actinomycosis, probably of dental origin, ended fatally as an actinomycotic meningitis. Two cases of echinococcus cyst occurred in

young boys, showing eosinophilia and a positive reaction of the skin for anaphylaxis. The test proved valuable in cases of echinococcus disease elsewhere in the body.

In the group of neoplasms (92 cases, table 2), the tumors of the optic nerve deserve special mention. While the first 4 cases, which I reported in 1922, were gliomas, the last 2 originated from the nerve sheath, and, as Cushing and Bailey described them, were true meningiomas, but they used to be designated fibro-endotheliomas. They were all characteristic, occurring at an early age, with a loss of vision that preceded the axial exophthalmos, a progression toward the brain, but never an invasion of the eyeball.

Among the malignant growths, the epibulbar neoplasms originate in the eyelids, lacrimal glands or conjunctiva. The periorbital tumors originate from the sinuses, extending into the orbit. In the group of sarcomas, the bulbar melanomas and gliomas are more malignant than

TABLE 2.—*Diagnosis of Orbital Neoplasms; First Surgical Clinic, University of Budapest, 1914-1930*

A. *Benign*		B. *Malignant*			
		Epithelial		*Mesenchymal*	
Fibroma	2	Epibulbar	14	Bulbar (melanoma and glioma)	23
Osteoma	5	Periorbital	3	Retrobulbar (round and spindle cell sarcoma)	23
Ganglioneuroma	2	Orbitoparietal	2	Orbitoparietal (osteogenic sarcoma)	12
Optic nerve tumor (glioma, fibro-endothelioma, fibromyxoma)	6	Total	19	Total	58
Total	15				

the retrobulbar sarcomas. The latter, if they are radically removed and irradiated, metastasize very late; much more malignant are the intraocular growths, of which the gliomas kill by continuous extension through the optic nerve, and the melanomas by late and distant metastases. The most malignant, however, are the lymphatic growths behind the eye, namely, the chloromas, of which I had 1 case. The osteogenic sarcomas of the orbital walls, if they can be removed radically, do not metastasize locally and hence should be attacked even if they are extensive, because distant metastases represent much less suffering.

SURGICAL TREATMENT

The operations performed in the first 109 cases have been published in 1922. I have no detailed data on the additional cases, but the methods have remained the same (table 3).

Most orbital operations, with the exception of those on children or in the presence of acute spreading infections, were done under local anesthesia. Premedication the night before and an hour before the operation was given in the form of barbital or one of its derivatives.

One-sixth grain (0.01 Gm.) of morphine was given subcutaneously half an hour before the operation. Orbital anesthesia was accomplished by the method of Hertel, medial lateral and inferior orbital injections being made, the ciliary ganglion being blocked, and, in addition, if a resection of the lateral wall was planned, a copious infiltration of the posterior surface of the frontosphenoidal process of the malar bone was given. I have used a block of the gasserian ganglion only once; the anesthesia was perfect, but the method is too complicated and not entirely devoid of danger, so that its routine use cannot be recommended.

All intra-orbital explorations are started as a transpalpebral orbitotomy, as described by Herman Knapp and Rollet. The incision is made in the unshaved eyebrow, and after splitting the tarso-orbital septum, the periorbita is exposed and incised or lifted up from the bone as the case may require. A number of orbital lesions can be explored

TABLE 3.—*Operations for Orbital Lesions;* First Surgical Clinic, University of Budapest, 1914-1930*

Type of Operation	Number of Cases
Transpalpebral orbitotomy	35
Transpalpebral-conjunctival orbitotomy	1
Resection of lateral wall	55
Approach through medial or inferior wall (sinus operations)	70
Approach through superior wall (craniotomies)	6
Exenteration of the orbit	74
Orbitosinusal exenteration	10
Ligation of common carotid	8
Ligation of ophthalmic varices	4
Total	263

* First 109 cases reported in 1922.

and removed in this way, without having to resect any part of the bony wall, so that every osteoplastic resection should start as a simple orbitotomy, and only if this proves insufficient should the bony wall be resected. If the tarso-orbital septum is carefully reconstructed and the skin is sutured as a second layer over it, the ptosis is only of a few weeks' duration; even if one cannot suture the wound, as after the incision of abscesses, the scar will pull up the eyelid with hardly any permanent deformity.

The approach through the lateral orbital wall, usually referred to as the Krönlein operation, has been somewhat modified (fig. 1). The skin incision is that of Kocher, which runs parallel with the facial fibers.

It starts in the temporal half of the eyebrow, follows the lateral margin of the orbit as far as the external canthus and then bends over to the malar bone, directed toward the lobe of the ear. The orbital ridge is then freed from the periorbita medially and from the temporal muscle laterally, after splitting the temporal fascia. Instead of removing the whole lateral wedge of Krönlein as far as the infra-orbital

fissure, I chisel off the lateral orbital margin, leaving a ridge about 0.5 cm. wide in connection with the soft tissues. The chisel is a very narrow, oblique instrument and is inserted well above the zygomaticofrontal suture line, higher than in the original Krönlein operation. A wide spatula protects the orbital contents. When the bony margin is well retracted, the rest of the lateral orbital wall is chipped off with a Luer forceps according to need. The periorbita is then incised above or below the external rectus muscle, and the muscle is either retracted or deliberately severed, if more room is necessary. The rest of the operation is atypical, care being exercised not to injure the optic nerve and, what is more easy to do, the ciliary ganglion. This

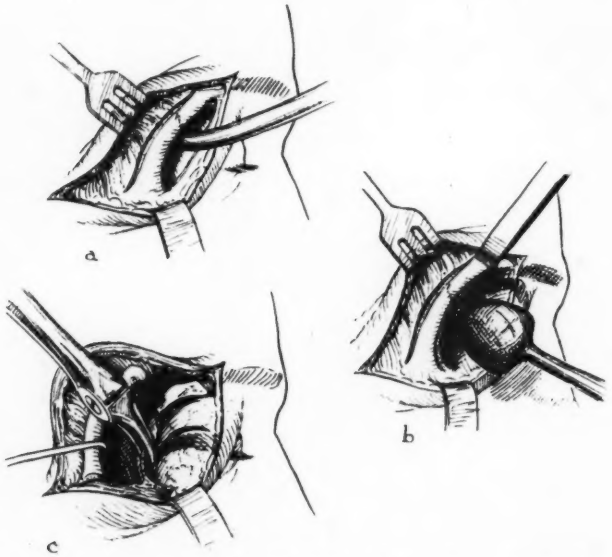

Fig. 1.—Resection of the lateral wall of the orbit: In *a*, Kocher's incision starts in the temporal half of the eyebrow, follows the lateral margin of the orbit and bends over to the malar bone directed toward the lobe of the ear. The orbital margin is freed from the periorbita and from the temporal muscle, by splitting the temporal fascia. In *b*, a narrow chisel is inserted well above the zygomaticofrontal suture. The orbital contents are protected with a wide spatula. In *c*, the bony margin is retracted with the soft parts, and the rest of the lateral wall is chipped off with a curved Luer forceps. The periorbita is incised above or below the external rectus muscle.

approach combines the simplicity of H. Gifford's "definite resection of the lateral orbital wall" and the cosmetic advantage of retaining the lateral bony margin. It is far simpler than Krönlein's wedge-shaped resection, in which the wedge, at least in my experience, does not always fit snugly when it is replaced, and the extensive bone resections of Kocher and Czermak.

I have records of 2 cases of permanent paralyses of the external rectus muscle; also of 6 cases in which a more or less extensive corneal anesthesia was present with a paresis of the pupillary sphincter, in which cases the ciliary ganglion must have been injured. In extensive growths injury to this pink nodule, 2 mm. long, lying in the sagittal plane between the external rectus muscle and the optic nerve, is not surprising.

The approach through the paranasal sinuses is indicated when the original cause of the orbital disease is in the sinuses. The frontal and ethmoidal sinuses have been exposed by Killian's method, maintaining the supra-orbital margin and draining through the nose. It is wise to remove the middle turbinate a day or two before the operation. The endonasal approach in acute pyogenic infections that have spread to the orbit does not seem the proper solution. The maxillary antrum was exposed according to Denker's method. If the inferior orbital wall is invaded or must be removed, a flap of the temporal muscle helps to maintain the eyeball in place. I did this in two instances, with good cosmetic results.

The approach through the superior orbital wall was resorted to when the growth invaded the cranial cavity and when it originated there. Thus six craniotomies had to be performed. The possibility of having to expose the meninges to an infected nasal sinus must be kept in mind.

Exenteration of the orbit was practiced according to a modification that I described in 1921.[4] If possible, the eyelids are conserved, and the incision is first made through the external and internal canthus. Very often, however, the malignant growth has infiltrated one or both eyelids, and they must be sacrificed with the orbital contents. In either case the incision is made straight down to the orbital margin, the periorbita is separated with a periosteal elevator, and the pedicle is cut as far back as possible. The ophthalmic artery and vein are tied with a suture ligature, and the whole field must be quite dry. Next an opening is made through the lamina papyracea of the medial orbital wall into the nose. A small flap of mucous membrane is laid into the orbital cavity, and a cigaret drain is inserted through the nose into the orbit. If there is oozing, a light iodoform pack may be used. The orbit is closed entirely from the front by interrupted sutures (fig. 2). If the lids are available, they are used, after the eyelids and tarsus have been trimmed off; if there is not enough skin, a frontal flap is brought down with a temporal pedicle and sewn into place (fig. 3). The defect on the forehead is covered with a skin graft. The drain or pack is removed in forty-eight hours, and the orbital cavity can be irrigated daily with hydrogen peroxide to maintain adequate drainage.

4. de Takáts, Géza: Plastik der Augenhöhle nach ihrer Ausräumung, Deutsche med. Wchnschr. **47**:1132, 1921.

The operation proves most satisfactory, in that it turns the orbit into a closed paranasal sinus, and the prolonged granulation and epithelialization of the orbital cavity are avoided. For some time there is a flapping of the skin flaps on blowing the nose, but later the skin is bound down to the bony ridge and remains immobile. The patients were usually discharged on the tenth day, a great saving of time in the hospital.

The orbitosinual exenteration described by Golovin was done in 10 cases. In extensive carcinomas of one or more sinuses, which invade the orbit, a radical extirpation of all invaded sinuses and the orbit is

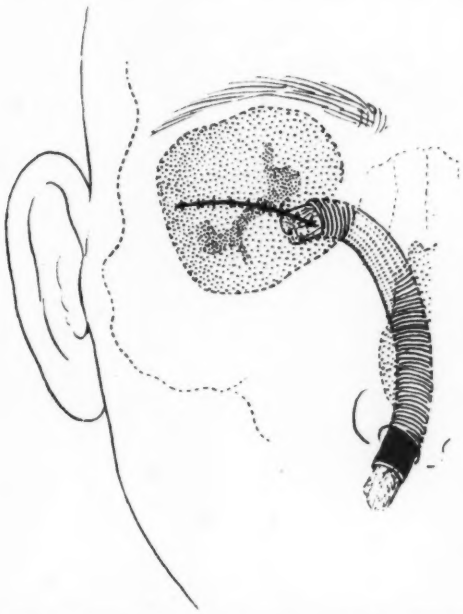

Fig. 2.—The orbital contents have been removed with the periorbita. Bleeding has been controlled by a suture ligature of the ophthalmic vessels. An opening is made through the lamina papyracea into the nose; a small flap of mucous membrane is laid into the orbital cavity, which is drained with a cigaret drain through the nose. The orbit is closed entirely from the front by suturing the eyelids with interrupted sutures. Thus the orbit has been turned into a paranasal sinus.

performed. Closure again is made in front, with adequate drainage to the nose.

Ligation of the common carotid, which was done for pulsating exophthalmos, is not without possible untoward reactions. In order to avoid cranial complications, the following safety measures have been employed: (1) The operation was preferably done on younger patients, in whom better collateral circulation could be expected; (2) intermittent elastic compression was applied several times daily, for two weeks; (3) when cerebral symptoms were absent after the artery had been clamped for

a few minutes, the artery was tied with a strip of fascia lata 1 cm. wide, an injury or folding of the intima being carefully avoided; (4) the internal jugular vein was tied at the same level, as ligation of a vein is known to diminish the percentage of gangrene in the extremities, when the main artery is tied. Thus the two most common causes of cerebral injury, namely, anemic softening through lack of collateral circulation and ascending thrombosis from the ligature, may possibly be diminished. Of the 8 ligatures, 6 gave excellent results, 1 was of little effect, and in 1 case, that of an older woman, hemiplegia developed,

Fig. 3.—When the eyelids are not available for plastic closure of the orbit, a frontal flap is brought down with a pedicle that contains the temporal artery. To ensure a snugly fitting flap, the skin of the temporal region is undermined and the flap is brought over under the skin of the temple. The defect on the forehead is covered with a thick Thiersch graft.

which subsided in two weeks. This was one of the earlier cases, when preliminary compression and ligation of the vein were not employed.

The ligature of orbital varicosities was practiced after a resection of the lateral orbital wall in 4 cases of intermittent exophthalmos. Two of these patients gave the history of ophthalmic phlebitis and really represented a "milkleg" of the orbit, with considerable venous and lymphatic edema. Two other cases had a history of trauma. The differentiation from true vascular anomalies, however, is hardly possible. In the 2 cases of orbital edema the intermittent exophthalmos was hardly benefited; in the other 2 cases there was a perfect result.

COMMENT

The classification and surgical treatment of orbital lesions, as presented here, illustrate the fact that the surgeon who undertakes the exploration of the orbit must be ready to undertake an intracranial or a rhinological operation, and that a simple orbitotomy may have to be continued by a bone resection and extended to an exenteration. The close proximity of the cranial and nasal cavities, the variety of delicate structures and the important mechanisms contained behind the eyeball produce most interesting diagnostic and surgical problems.

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CORRECTION

In the article entitled, "Abuse of the Theory of Focal Infection," by Dr. O. R. Lourie in the July issue (**8**:24, 1932) the first word on page 27 should be "replaced" instead of "placed."

Clinical Notes

EYE AND EAR IN "SEASICKNESS"

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Chance reading of a paper by Lebensohn,[1] written two years ago, suggests publication of some observations I made a number of years ago and never reported. Lebensohn wrote:

> From these experiments, one can definitely conclude that car-sickness is due to labyrinthine stimulation rather than to optic nystagmus. Optic nystagmus is easily tolerated and is not accompanied by depressive gastric phenomena. The impulses from the labyrinth, however, are depressive. . . . Though car-sickness is based on labyrinthine irritation, it is nevertheless true, I believe, that errors of refraction and of muscle balance predispose a person to this malady.

The observations I here report indicate that Lebensohn's statements are not adequate. The matter is more complex than he indicated.

My observations were made on myself, a good subject because I am unusually prone to seasickness.

I have made numerous experiments, probably twenty or more, off and on for forty years. The subject of the experiment stands quiet at a distance of from 4 to 15 feet from a long freight train passing at moderate speed or faster. He looks directly and steadily at the cars, endeavoring not to follow individual cars along the track, but to look fixedly at the train as it passes at a right angle. In oftentimes less than a minute nausea occurs, and half a dozen times the experiments were carried to the point of bringing about reversed peristalsis of the intestine, but only a couple of times were they carried to the point of actual vomiting. Several times the experiment was carried out as follows: first, the subject looked at the train until nausea had just begun, then looked away for a minute or more, until the nausea passed, and then looked again at the passing cars, with the result that nausea returned, and this time it came more promptly than before. The experimenting was never carried to the point of extreme dizziness, such as would cause staggering, though some dizziness is remembered. The subject could not observe his own eyeball motions and so cannot say whether slight motions occurred in spite of the endeavor to keep a fixed stare.

This experiment followed an informal discussion of a suggestion, made by me, that tobacco sickness might be due to disturbance of the semicircular canals. The subject of the experiment had smoked tobacco occasionally between the ages of 8 years and 12 years, enough for him to acquire tolerance for the poison. After twenty years of complete abstinence, and following the discussion mentioned, the subject took a fairly strong cigar (a black "Harvard"), lay down to read and, keeping quiet on his back, smoked it all. Having no sensation of dizziness or

1. Lebensohn, James E.: "Car-Sickness," Arch. Ophth. **4**:342 (Sept.) 1930.

nausea, he concluded that his immunity, due to youthful indulgence, must still be persisting. But on rising to throw the cigar stub out of the window, wave after wave of extreme dizziness passed over him. He returned immediately to the bed, not without some staggering, but before the reversed peristalsis which had begun had reached the point of vomiting. He lay again perfectly quiet on his back until all nausea and dizziness passed away, say three minutes or less. Then for a half or three quarters of an hour or so, these further experiments were tried. The head would be moved, rotated, with a quick jerk, but through the least possible arc, about a third or a quarter of an inch, successively in the planes of the three semicircular canals, with intervals of quiet between the motions. The slight but quick jerk was, in each case, followed by a wave of dizziness which passed off after less than a minute of quiet. More of a rotation, or two or three slight, quick jerks in succession, brought nausea, and this was repeatedly carried to the point of starting reversed peristalsis, but was always stopped short of vomiting. There can, however, be no doubt that further stimulation of the sort would have produced vomiting. The subject then slept through the night, having no further inconvenience. In sleep during the night no dizziness, nausea or vomiting was noticed. There may have been no motion such as would have caused them.

Indications from these experiments and some suggestions follow:

1. Eye stimulation, apparently stimulation of the retina with changing images and without much, if any, motion of the eyeball, can produce nausea, dizziness and, if persisted in, vomiting. This seems in disagreement with Lebensohn's conclusion, though he emphasizes eye motions rather than retinal images.

2. Tobacco-poisoning renders the semicircular canals hypersensitive, so that even very slight rotation causes dizziness, nausea and, if persisted in, vomiting. The stimuli in these experiments were far too slight to produce noticeable results on normal balancing organs.

3. Overstimulation of normal balancing organs probably is an efficient cause of dizziness, nausea and vomiting, if exceedingly slight stimulation of balancing organs hypersensitized by tobacco can produce these effects.

4. Ear symptoms (dizziness) usually precede stomach symptoms (nausea) in the onset of seasickness.

5. Excessive motion, as on shipboard or on a railway train, can produce seasickness during sleep or in the dark, indicating that retinal stimulation is not necessary to produce it.

6. While either eye stimulation or stimulation of the semicircular canals can produce seasickness, it is probable that generally both types of stimulation are involved. Many times, when beginning to feel a little sick on shipboard, I have inhibited (rarely), or usually merely delayed, extreme nausea by looking steadily at the horizon line. I have the impression that the procedure is more successful when the subject stands up. Looking at the horizon when sitting steadies the eyes. Doing so when standing steadies the whole body, including the head, by balancing against the motions of the ship.

7. The matter is still more complex, for there seems to be sufficient indication that previous stomach disturbance, indigestible food or too much food may increase liability to seasickness under proper stimulation.

A COMBINED NEEDLE AND CAPSULE FORCEPS

JOSEPH L. MCCOOL, M.D., SAN FRANCISCO

Many of the instruments used in ophthalmic surgery were devised to meet emergencies; this is especially true with regard to cataract operations. I know of no operation in surgery in which the need for prompt action is greater than in this one, the difference between success or failure depending on the instant availability of the proper instrument.

Whether the operator uses the capsulotomy or intracapsular method of extraction, impending or actual loss of vitreous is the gravest complication with which he has to deal. If the hyaloid ruptures and vitreous presents itself before the lens engages in the wound, all pressure on the globe, whether voluntary or involuntary, should at once be removed. To meet this emergency it is necessary, therefore, to extract the lens rather than to express it, and numerous instruments have been devised to effect this delivery. Wire loops, either plane or barbed, Reissinger sharp double hooks, scoops of various shapes, and finally the smooth spatula used by Colonel Henry Smith in his intracapsular method, have all been used to meet this emergency according to the individual operator's fancy.

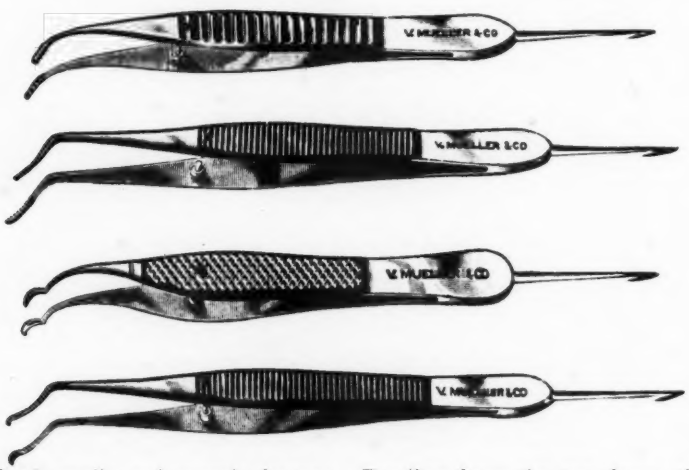
Colonel Smith uses the spatula to hold back the vitreous, at the same time utilizing its smooth anterior surface as an inclined plane over which the lens slides in its delivery. Dr. William Fisher of Chicago modified this spatula by having a very sharp needle placed in the other end. I have used this instrument ever since it was devised by Dr. Fisher, and have found the needle extremely valuable. I have not used the Smith Indian intracapsular operation for over fifteen years; consequently, with the technic I now use, at a certain stage in the extraction of the lens the assistant was obliged to exchange the forceps for the needle. Obviously, the need for the substitution came at a time when every second counted.

In order to eliminate the change of instruments, I had an instrument maker put a long, sharp needle in the end of my capsule forceps. For those operators who still use the cystotome, this instrument will have no appeal. The capsule forceps, however, are used so universally, both in the capsulotomy and intracapsular operation, that it seemed to me that this addition to the instrument might find favor with those who use them.

Those I use are the new model Ewing and Vail forceps, but the needle could be placed in the Kalt forceps or any of its modifications. While the Ewing and Vail forceps are primarily capsule forceps and have for their principle object the removal of a large portion of the anterior capsule, in a surprisingly large proportion of cases, by using the forceps to fix the lens and making counterpressure below, the zonule will rupture and the lens will tumble. If the zonule proves to be more resistant than the capsule, the forceps will remove a sufficiently large bite of the latter to ensue a clear pupil; if the zonule ruptures, the lens

revolves around its transverse axis and the lower border presents itself in the wound. The capsule forceps may still retain its hold on the unruptured capsule, but despite the fact that the cornea is tucked between the lens, it occasionally refuses to budge. At times it is not out of the wound far enough to permit rotation by the concavity of the expression hook; in that event we are compelled to maintain its upright position by gentle pressure with the tip of the expression hook while we release our grasp on the capsule, reverse the forceps, transfix the lens and rotate it out of the wound.

In the last instrument I had made, barbs were placed near the point for traction purposes, if necessary. In case the capsule ruptures early and the forceps are removed, the technic need not be varied, for in a



Combined needle and capsule forceps. Reading from the top down, the needle is shown attached to the forceps designed by the following men: Ewing, Vail, Findlay and Kalt.

number of cases it is just as easy and safe to tumble the nucleus by gentle pressure below, applied correctly, as it is to deliver in the orthodox manner provided you have the needle to assist in the delivery. However, in the upright delivery one can still find the instrument useful by using the closed end of the forceps for counterpressure and the needle for any emergency that may arise.

The instruments are made by V. Mueller and Company of Chicago.

A MODIFIED MONOCULAR LOUPE

EMANUEL KRIMSKY, M.D., BROOKLYN

One of the difficulties confronting the ophthalmologist in the use of the monocular loupe is in obtaining a sufficient amount of condensed light at a suitable focal distance. Besides the fact that one hand is occupied in holding the condensing lens, the source of illumination

may not always be close or conveniently at hand. In order to overcome these shortcomings it occurred to me that if the light source and condenser could be combined as a unit and this in turn be attached or fastened to this loupe, a satisfactory and reliable source of illumination might be effected to assist the operator in his observations. In the construction of such an instrument the following features seemed essential: (1) centralization and proper angulation of light at, say 45 degrees, (2) firmness, (3) lightness, (4) compactness and simplicity of operation, (5) decreased cost and (6) proper quality and quantity of illumination.

REQUISITES

Centralization and Proper Angulation of Light.—This was determined by projecting an imaginary vertical line through the center of the loupe which would fall on some fine print at the maximum focal distance for the loupe used (10 × Zeiss) and which was found to be 19 mm. from the under surface of the lens. From this imaginary centralized focal point another line was projected 45 degrees upward and outward to represent the angle through which the light carrier would be tilted.

In this photograph of the instrument described, wing-nuts are not shown. Instead there are ordinary screw-heads.

Firmness.—The device which locks both the loupe and light carrier is a combination bracket and clamp holder; the former is 3 mm. thick and operated by a single wing-nut which is deftly drilled into the framework of the loupe proper and serves to attach the bracket securely to the loupe; in addition, the bracket is reenforced by a hooklike prolongation about its head. This bracket is continuous with a clamplike device operated by two wing-nuts and joined to a thicker (5 mm.) flap of brass which is notched at an angle of 45 degrees to hold this light carrier securely.

Lightness.—The weight of my 10 × Zeiss loupe is 33 Gm. The light carrier used in my model is part of the old Wappler ophthalmoscope and weighs but 10 Gm. The bracket-clamp unit weighs 24 Gm., and when these are operated as a combined unit the 67 Gm. of weight does not seem appreciably heavier than that of the loupe alone. The light carrier is connected to the battery by means of a lighting cord to economize on weight and ease of operation.

Compactness and Simplicity of Operation.—By a simple process of unscrewing, this bracket-clamp may be easily disassembled and the loupe and light carrier may be separately folded away. Lateral to the

heads of each of these clamp-screws, notches have been provided which would enable the operator to lateralize the source of light if he chose to do so. The light carrier may be brought nearer or further away from the focal point of the loupe through this 45 degree radius by simple manipulation of this clamp.

Decreased Cost.—While the construction of such a unit does not entail much expense, especially to one who already owns a light carrier of this type, there is one feature that could be improved on from the standpoint of cost, namely, the 2.5 volt lamp, which costs $1.30.

Proper Quality and Quantity of Illumination.—The intensity of illumination is satisfactory. By simple manipulation of the condenser, one may obtain a homogeneous circle of light or a slit of varying size—an advantage never to be secured with the clumsy method of using a condenser in the other hand.

REFLECTING PRISM-LENS FOR SLIT-LAMP MICROSCOPY OF THE RETINA

JOHN N. EVANS, M.D., BROOKLYN

Ophthalmologists have had a desire to study the retina by means of the slit-lamp and corneal microscope. They have come to realize that the use of the contact glass as an auxiliary for this purpose will not become popular.[1]

The following is the description of a simple and inexpensive attachment for the standard slit-lamp which apparently solves the problem.

A planoconvex condensing lens is mounted on a rod support, as indicated by the accompanying photographs. To one face of this lens and at its optical center a small right angle prism is mounted in such a way that a pencil of light entering one of its faces will be bent 45° (totally reflected) and will emerge coincident with the optical axis of the lens (fig. 1). The lens need not be corrected for spherical or chromatic aberration.

A small clamp is next secured (one of a standard type of clamp used in the physiologic laboratory is satisfactory) by which the mounting carrying this lens-prism combination is attached to the end of the slit-lamp arm. Adjustments are then made so that the beam of the slit-lamp

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From the Research Department of the Brooklyn Eye and Ear Hospital.

1. The recently described Friedenwald hand ophthalmoscope (Tr. Am. Ophth. Soc. **26**:381, 1928) is adapted for the examination of the retina, and the illuminating system may be so adjusted as to produce an optical section of the retina. No contact glass is used.

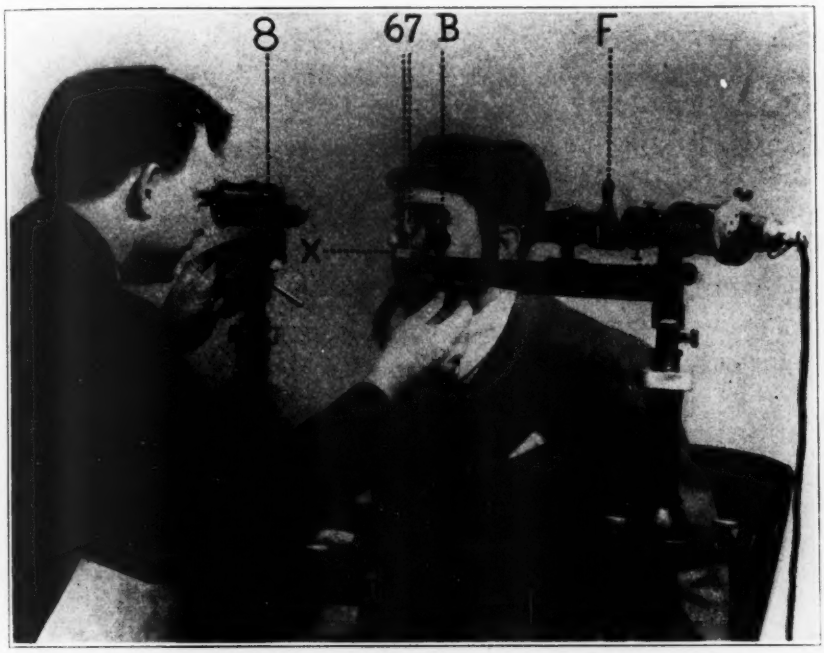


Fig. 1.—Photograph illustrating the technic to be used and the mounting of the reflecting prism-lens. Clamp *X* holds the lens carrier at the extreme end of the slit-lamp arm. The lens (6) is so adjusted as to bring the small prism (7) into the beam emerging from the aplanatic lens (*B*) of the standard slit-lamp. The prism then deflects the beam at right angles through the patient's pupil where it can be accurately focused on the retina by adjusting the lens (*B*) in the usual manner. The observer, looking through but one tube of the corneal microscope (8) (ocular and objective removed to line up the microscope) begins at a distance of about 30 cm. from the patient and locates the aerial image which is situated somewhere between the microscope and lens (6). The low power shows a sharp optical section in the retina, but the high power develops detail in but a small portion of this optical section. High powers must be carefully adjusted as the depth of focus is shallow. *F* indicates the groove in which an orange filter may be placed (the tube reversed to make it visible in the photograph). This filter removes the violet, blue and green, leaving only the closely related red, orange and yellow of the spectrum which focus in about the same plane. Note the great distance between the microscope and reflecting prism-lens. It is important not to destroy the polish on the optically acting faces of the prism. There is danger of destroying its reflecting properties if cement is applied directly to the glass. In order to avoid this it is necessary either to silver the reflecting face before cement is applied or to mount the prism on the image forming lens by means of a minute metal clip which will be attached to the sides of the prism and cemented to the face of the lens.

can enter the prism and be deflected through the dilated pupil. This obviously requires that the slit-lamp arm be turned at right angles to the subject's visual line. The reflecting prism lens is set as close to the patient's eye as possible, and so that the beam can be seen entering the pupil. The corneal microscope is adjusted before this lens at about 30 cm. distance from it. The angle formed by the two tubes of the corneal microscope is so great that it is impossible even with the dilated pupil to see a point on the retina through both at one time. The objective and ocular of one of the tubes is removed and the microscope manipulated until the examiner can see the fundus reflex in the pupil with a shadow of the prism at its center. The lowest powers of ocular and objective are then slipped into place and the microscope carefully

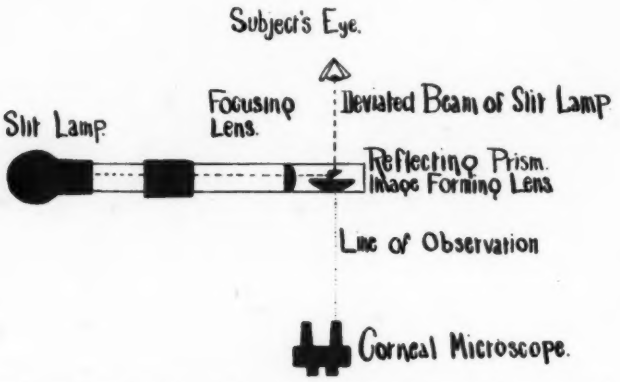


Fig. 2.—Diagram illustrating the relation of the subject, the viewing apparatus and the observer. The dash line represents the slit-lamp beam impinging on the face of the reflecting prism mounted at the center of the image forming lens. This lens forms an aerial image which is magnified by the corneal microscope. The corneal microscope, therefore, has the same relation to the image that the plus 3 or 4 lens does which is placed in the ophthalmoscope in indirect ophthalmoscopy.

focused. An optical section of the retina will then be visible. Its magnification will be considerable. The highest powers can be used, but as the focus is fine and the magnification enormous, one must learn to interpret the picture in order to appreciate detail.

Monochromatic light would be ideal for slit-lamp microscopy of the retina, but it does not seem practical to use filters or a spectroscope with the standard slit-lamp as the loss of light is too great. An orange filter is perhaps the nearest practical approach to the ideal.[2]

2. Brigham Colored Sheet Gelatine no. 135, Charles Products Company, 78 Cliff Street, New York, has been tested spectroscopically and found most appropriate.

The principle involved may be looked on as simply that of indirect ophthalmoscopy in which the reflecting prism lens [3] attachment is the image-forming system and the corneal microscope the magnifying system which enlarges the aerial image.[4] The appended illustrations should suffice to make clear any discrepancies of this description. Any competent optician should be able to construct the device at trifling expense.

23 Schermerhorn Street.

3. There is no image of the prism as it is placed within the focus of the image forming lens.

4. Application of the "reflecting prism-lens" to a hand instrument for ophthalmoscopy is to be described in a separate report. A reflecting prism may be similarly attached to a camera lens for the illumination of such cavities as the mouth. The same principle may be used for the projection of pictures.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

THE ANATOMY AND THE MECHANISM OF THE LACRIMAL SAC. A. FAZAKAS, Klin. Monatsbl. f. Augenh. **87**: 73 (July) 1931.

A perfect operation to reestablish the disturbed function of the tear sac, in the author's opinion, cannot be found until the hitherto unknown normal mechanism of tear conduction becomes known. Fazakas, therefore, studied this question in operative cases by analysis of fistulas by roentgen and manometer examinations and by macroscopic and microscopic research. He found that during operations the dilatation of the tear sac is produced by the function of Horner's fibers, while the batting of the lids closes the puncta. This was observed also in cases with closure of the tear points. Simultaneous dilatation and contraction of the lumen of the tear canals as well as of the saccus could also be noticed during operations and in cases with congenital or acquired fistulas of the canaliculi. The theory of this mechanism is discussed in detail, and experiments with a manometer, which was introduced into the fistula, are described. The function of the tear sac is lacking when it is enlarged due to closure of the ductus. Roentgenograms allow conclusions regarding the physiology of tear conduction, but all studies of the mechanism are rendered difficult because the iodized poppy seed oil 40 per cent leaves the duct too rapidly. Negative results with the manometer and the absence of changes in the shadows of the iodized oil during all phases of the batting of the lids are indicative of the impossibility of reestablishing the mechanism in any other way than by extirpation of the tear sac. Positive results promise a possible restitution of the tear conduction by means of dilatation, or by the formation of a new passage from the tear sac into the nose.

K. L. STOLL.

Color Sense

DALTONISM. RAUL ARGAÑARAZ and ESTEBEN ADROGUÉ, Arch. de oftal. de Buenos Aires **6**: 649 (Nov.) 1931.

After reference to Eingelking's investigations and conclusions presented at the last International Congress of Ophthalmology, and to the necessity of standardizing the tests to which the personnel of railroad, naval and aerial concerns should be subjected, the author stresses on the production of Rayleigh's equation, obtained by comparing a homogeneous light with a wavelength of 0.589 microns with two lights of 0.535 and 0.670, respectively, or the yellow sodium light with a mixture of the red lithium or green thallium lights. This is only obtainable, for clinical purposes, by means of Nagel's anomaloscope, which permits the recognition of protanopia, deuteranoia and trichromatopia and at the same time determines the amplitude of the focus. Eingelking proposes

that only those persons be considered normal who present an equation of from 57 to 65, measured by the anomaloscope, with a focus amplitude of not more than 6 lines.

The authors next explain their diagrammatic rendering of the equation. Of 1,500 persons examined by them, 90 (6 per cent) showed anomalies in color perception, which could be classified as follows: protanopia, 27; deuteranopia, 25; protanomalopia, 8; deuteranomalopia, 14, and weakness for colors, 16. Investigations conducted in connection with the influence of inheritance have not progressed sufficiently to permit any definite conclusions.

C. E. FINLAY.

Conjunctiva

THE CLINICAL RELATIONS BETWEEN TRACHOMA AND TUBERCULOSIS. T. BARDANZELLU and E. TROVATI, Arch. di ottal. **38:** 621 (Dec.) 1931.

One hundred consecutive patients with trachoma were given a complete physical examination, including tuberculin tests, roentgenograms and fluoroscopic examination of the chest. The chest findings are classified as is the incidence of each group in patients with the various types of trachoma. Seventy patients showed positive roentgenoscopic findings, and fifty-eight positive skin tests. Only six showed active tuberculosis, the majority showing the glandular or latent pulmonary forms. These were especially common in cases with the more severe types of trachoma showing corneal involvement, and in children. The authors believe that tuberculous infection predisposes a person to the development of a severe follicular reaction of the conjunctiva when it is exposed to irritants such as bacteria of various kinds. This, they think, helps to explain the fact that certain persons develop trachoma by contagion, while others similarly exposed do not. They evidently do not consider trachoma as a definite disease due to a specific agent.

S. R. GIFFORD.

PURULENT CONJUNCTIVITIS OF THE NEW-BORN. ANGEL TORRES, Arch. de oftal. hispano-am. **32:** 65 (Feb.) 1932.

The author considers this name, purulent conjunctivitis, originated by Mackenzie and Wecker, as most appropriate. He considers the disease fairly frequent. In 965 children treated in the eye department of the Niño Jesus Hospital since 1926, 175 had purulent conjunctivitis.

The disease may be contracted in utero, at birth or after birth. The first group is usually attributable to a contamination of the amniotic fluid during vaginal examinations, but the author agrees with Haussmann and Kraus that the infection may occur through the lymphatics. The second group is the most numerous, the gonococcic origin not being as common as supposed. The 175 cases observed were caused by: gonococcus alone in 39 cases; coli bacillus and streptococcus in 26, pseudo diphtheria bacillus in 4, pneumococcus, streptococcus and coli bacillus in 35, coli bacillus and Koch's bacillus in 19, streptococcus alone in 7 and nonmicrobic origin in 45 cases. One hundred and twenty-five of the 175 mothers gave a history of prior vaginal discharge, in 98 of which it disappeared after birth. Etiologic influence of the duration of labor is shown by 10 cases occurring after two hours labor, 36 after

three hours, 53 after four hours and the remaining 76 in labors of over five hours.

One hundred and twenty cases occurred in primiparas, 37 after a second labor and the remaining 18 occurred in multiparas.

The cases occurring after birth are due to faulty method of washing babies, to prophylactic measures employed and to infection by caretakers.

In connection with "inclusion conjunctivitis," the author refers to Lussmanns, who found Prowazek bodies in about one half the cases.

The author refers to harmful action of the employment of too concentrated silver nitrate and advocates its substitution with mercuric chloride, 1 : 4,000; mercuric cyanide; strong silver protein, 10 per cent; albargine; mild silver protein, 5 per cent; silver acetate, 1 to 2 per cent. When mothers present a vaginal discharge, he recommends douching with a solution of potassum permanganate and mercuric chloride before labor.

In the treatment of the eye, he limits the application of silver nitrate to the first four or five days and washes the eyes with a 1 : 3,000 solution of potassium permanganate followed by the instillation of drops of mild silver protein every two hours. When there is corneal ulceration, the eyes are flushed with solutions of potassium permanganate or mercuric chloride and an ointment is applied. Prolapse of the iris is treated with atropine or pilocarpine according to the situation of the perforation. When the pneumococcus is present, ethylhydrocupreine hydrochloride is used locally.

C. E. FINLAY.

THE ETIOLOGY AND THERAPY OF VERNAL CATARRH. P. WEINSTEIN, Klin. Monatsbl. f. Augenh. **86:** 802 (June) 1931.

The author discusses the numerous theories supposed to explain the genesis of vernal catarrh. He then refers to recent experiments which indicate the possibility that vernal catarrh is an anaphylactic phenomenon, as is bronchial asthma, hay fever and urticaria. He examined thirty patients suffering from vernal catarrh. They were subjected to tests for calcium content of the serum after the method of Kramer and Tisdall, for alkali reserve after the method of Van Slyke, for serum refraction and for intracutaneous reaction to a large number of pollens. Calcium content of the four patients examined was increased; the alkali reserve slightly decreased; the intracutaneous reaction was positive in eight cases, and positive, but complicated with another hypersensitiveness in another patient. Positive results were also obtained in a number of controls with trachoma. Weinstein arrived at the conclusion that in vernal catarrh the disposition and the shifting of the acid-base balance play an important rôle. The combination of vernal catarrh with bronchial asthma and the hypersensitiveness to certain pollens suggests that vernal catarrh belongs to the group of anaphylactic diseases. Therapy consisting of endocrine preparations from the parathyroid, suprarenal gland, ovary and testicle, also calcium solution and vitamin D, are indicated on account of dysfunction of the nervous system and the endocrine glands.

K. L. STOLL.

Congenital Anomalies

A New Case of Congenital Absence of the Lateral Movements of One Eye (Stilling's Syndrome). H. Coppez, Arch. d'opht. **49:** 18 (Jan.) 1932.

Many cases of this kind have been described under different titles. Stilling was the first to describe a typical case, and it is for this reason that the author believes they should be classified under the designation of Stilling's syndrome. A typical case is described well illustrated with photographs. The fundamental condition is congenital absence of the lateral movements of one eye, almost always the left. The other symptoms are less constant and are, in the author's opinion, secondary. Enophthalmos is frequent, and sometimes there is an actual retraction of the globe on adduction. Sometimes on adduction the eye turns up and disappears under the upper lid. Few cases have been seen in which the eye turned down on adduction. Narrowing of the palpebral fissure frequently accompanies retraction of the globe and is due to its displacement. The author believes that the best evidence at present indicates the condition to be the result of peripheral anomalies of the muscles and tendons and not to nuclear or functional anomalies as suggested by some other writers. Few cases have appeared in the French literature.

S. B. Marlow.

Congenital Aniridia. Rómulo Gil and C. A. Locatelli Rivas, Arch. de oftal. de Buenos Aires **6:** 642 (Nov.) 1931.

A case of bilateral congenital aniridia in a child 2 months old is reported. A narrow stump of the iris was adherent to the base of the ciliary body; the macular region did not show the usual light reflexes. The child grasped and followed readily objects presented. There was no history of inheritance or consanguinity. The report is supplemented by general remarks and observations on the present status of knowledge on the subject.

C. E. Finlay.

Microphakia. F. Gnad, Klin. Monatsbl. f. Augenh. **87:** 33 (July) 1931.

Both eyes of a girl, aged 5, showed congenital relative and absolute smallness of the lens, and megalocornea of 13 mm. in the left eye, and of 13.5 mm. in the right. The central portion of the anterior chamber of the left eye was obliterated by close apposition of the lens and cornea. There was no iridodonesis present. The anterior chamber of the right eye was deep; the pupil was crossed by numerous strands of the pupillary membrane and the iris and lens showed vivid tremulation. Under homatropine the pupil measured 8 mm., leaving a space of 1 mm. between the margins of the pupil and lens, in which a few frail zonular fibers were noticed. The fundi of both eyes were normal. Pilocarpine lessened the tension of the right eye but caused pain, apparently because the iris was pressed against the cornea during the miosis. The tension in both eyes was reduced by extraction of the lenses under special precautions. The lenses were clear, of equal size and of spheroid shape. The correction in both eyes amounted to 14 convex diopters with 4 diopters cylinder; this would suggest that the globes were abnormally short.

K. L. Stoll.

Cornea and Sclera

BILATERAL SERPIGINOUS ULCER WITH SUPPURATION OF THE LENS AND POSTOPERATIVE PANOPHTHALMITIS, BASED ON CONSTITUTIONAL DEBILITY. P. VON PLANTA, Klin. Monatsbl. f. Augenh. **87**:39 (July) 1931.

A man, aged 54, had his left eye scratched by straw in 1924, and the right injured superficially in a similar manner in 1929. A serpiginous ulcer formed in the left eye, which could not be stopped by a Saemisch section of the cornea, and enucleation followed. This experience prompted the author to omit the section after Saemisch when a serpiginous ulcer developed after the injury of the right eye; the ulcer healed spontaneously after perforation of the cornea, leaving an anterior synechia and a white, tumescent cataract. Iridectomy, performed for secondary glaucoma three months later, was successful. However, the extraction of the cataract, a few months afterward, was followed by phthisis of the globe. This operation had been preceded by thorough bacteriologic examinations and lavage of the tear sac; a conjunctival flap had been made, and the lens and swollen cortical could be removed readily without entering the globe frequently with instruments. The conjunctival sac, free from bacteria prior to the operation, contained smeary pus, harboring pneumococci a few days later. Local treatment and parenteral injections of milk were of no avail. The extracted lens contained an infection, which, in the author's opinion, originated at the time when the perforation of the ulcer took place, and gave rise to the panophthalmitis, when the capsule was ruptured during the extraction.

Primary suppuration of the lens, caused by foreign bodies, was reported by Saemisch and Diel. The patient in Planta'a case presented an extraordinary low resistance to exogenous lesions, and extensive vitiligo, existing for many years, which suggested some endocrine disturbance. In closing, the author discusses the combination of vitiligo with diseases of the mind and nervous system, or with neuritides and neurotrophic conditions of the skin.

K. L. STOLL.

THE PHLYCTENULE AS A PHENOMENON OF IMMUNITY. F. SCHIECK, München. med. Wchnschr. **79**: 6 (Jan. 1) 1932.

Facts recently evolved, together with a comprehensive study of accumulated data, would point to the phlyctenule as a tissue immunity reaction.

A startling case in point is that of a patient, aged 35, with chronic gonorrheal urethritis, prostatitis and cystitis. At first there occurred a gonorrheal conjunctivitis due to direct contamination. A metastatic pleuritis intervened. Following this small round phlyctenules appeared on the upper corneal limbus. With treatment of the primary infection, the phlyctenules disappeared.

It has been noted that following instillations of "old" tuberculin into the conjunctival sac for diagnostic purposes, a local reaction may take place followed by the formation of phlyctenules. In both tuberculous and gonorrheal disease, the phenomenon may be explained as an antigen

—an antibody reaction. In order to prove this contention, W. Riehm's work with animal experimentation is cited. Rabbits were sensitized to horse serum, thus developing anaphylaxis, as shown by reactions to further injections of horse serum, such as shock and local inflammation of the skin. A typical phlyctenule developed on the limbus with the use of the antigen in the conjunctival sac.

The condition known as keratitis fascicularis, in which a "wandering phlyctenule" is noted, is explained as the reaction incited in the limbal vessels of the conjunctiva. Scrofulous diseases may have an underlying tuberculous basis, but the reaction is purely on immunologic grounds. Treatment should include methods of desensitization with a minimal injection of tuberculin, salt baths, sun baths and heliotherapy.

L. L. MAYER.

General

CONSERVATION OF EYEDROPS. K. MIKLOS, Brit. J. Ophth. **15:** 649 (Nov.) 1931.

The sterility of eyedrops is important because they are used after operations which open the globe and also after perforating injuries, when it is possible for micro-organisms to be carried with the drops into the internal part of the bulb, and thus cause dangerous infections.

As a result of the research to conservate eyedrops, the author arrives at the following conclusions:

Different esters of the para-oxybenzoic acid were tried in order to conserve eyedrops, and it was found that the most suitable of these is the propylester in 0.05 per cent concentration. The eyedrops thus prepared hinder entirely the growth of micro-organisms, and by the mild bactericidal effect those which enter the eyedrops die.

The eyes tolerate these prepared eyedrops well.

This method of conservation is recommended, because by it the physician does not have trouble in keeping the eyedrops pure.

Also, from an economical point of view, it is necessary to conserve eyedrops, because many of the alkaloids which are used in them are expensive.

W. ZENTMAYER.

A CURED CASE OF PHLEBITIS OF THE FACIAL, ANGULAR AND SUPERIOR OPHTHALMIC VEINS. PAULINA SATANOWSKY, Arch. de oftal. Buenos Aires **6:** 725 (Dec.) 1931.

A case is reported of phlebitis of the facial, angular and superior ophthalmic veins following a furuncle of the labial commissure in which an energetic antipyogenic treatment, associated with absolute repose and appropriate dieting, was followed by an excellent result. The author considers that in this case a septic embolus can be excluded, the lesion consisting of a directly propagated phlebitis which did not reach the meningeal veins. The antipyogenic treatment consisted mostly of injections of staphylococcus and streptococcus antigens in Ringer's solution, and later a colloid suspension of silver, with treatment of a complicating corneal ulcer.

C. E. FINLAY.

General Diseases

GRIP AND OPTIC NEURITIS. REPORT OF THREE CASES. E. JAEGER, Klin Monatsbl. f. Augenh. **86:** 812 (June) 1931.

Complications in the eyes, caused by grip, may occur in connection with encephalitis, or in general grip infection in form of optic neuritis. The latter exists only under the following conditions: negative Wassermann reaction, anamnestic absence of rheumatism, healthy nasal cavities, absence of sugar and albumin and normal blood pressure.

Jaeger reports three cases satisfyting these requirements. They show that toxic optic neuritis may develop several weeks after an attack of simple febrile grip, which had lasted about two to three weeks, seemed to have disappeared completely. The neuritis was very marked, although hemorrhages were generally absent; the vision was greatly reduced in one or both eyes. The prognosis was favorable even in severe cases. Women suffer from this form of neuritis more frequently than men. It is difficult to say whether the rarity of this neuritis in grip is due to the kind of the virus, or to the individual sensitiveness of the afflicted optic nerve. Rest in bed may suffice to effect a final cure, yet recovery was decidedly more prompt when methenamine was given, which the author injected intravenously in doses of 5, 4 and 3 cc. successively.

K. L. STOLL.

Instruments

AN ELECTRIC OPHTHALMOSCOPE. N. B. HARMAN, Brit. J. Ophth. **16:** 102 (Feb.) 1932.

Harman has adapted the parts of his well known reflecting ophthalmoscope to a self-contained electric fitting.

It has a range of 70 diopters from 30 to — 39 in steps of 1 diopter, yet it is no larger than a May's ophthalmoscope. The battery is a standard Ever Ready. The lamp is gas-filled and has a small coil filament. A fine control of the focusing lens movement is obtained by a focusing lens controlled by an external collar, so that the beam of light may be slightly divergent, convergent and increasingly divergent.

Four cells can be interposed between the light and the mirror, containing filters, red, green and "daylight," and a small hole diaphragm.

The handle is covered with fine sand, which gives a perfectly easy grip.

W. ZENTMAYER.

Lacrimal Apparatus

LATENT INFECTION OF THE LACRIMAL DUCTS. E. KALT and M. KALT, Ann. d'ocul. **169:** 1 (Jan.) 1932.

The authors conclude that latent infection of the lacrimal ducts generally originates from the conjunctiva. Its nature is not well known, but without doubt it is microbic, and it is more liable to occur in receptive persons, such as the aged. It corresponds to a pathologic entity, from which it is necessary to distinguish the ascending infections of nasal origin, which first obliterate the inferior orifice of the sac with subsequent ectasia.

The distinction between the two diseases, founded on clinical and pathologic anatomy, seems evident, and the authors think that a good title to be given to the first is dacryocystitis of follicular stenosis.

The diagnosis of latent infection is difficult in certain cases. The authors have found that permeability to injection is a valuable sign, certainly the best there is, provided the injection is performed by an operator who is accustomed to the work; it is not sufficient to say that it was done, but how. Even if the passage of fluid was free, the sac may have been gravely altered. Bacteriologic examination is a secondary matter and gives uncertain results.

S. H. McKee.

A Case in Contribution to the Etiology of Hypofunction of the Lacrimal Gland. K. Hauer, Klin. Monatsbl. f. Augenh. **87:** 79 (July) 1931.

Hypofunction of the tear gland with decreased moisture and secondary keratitis has been described by Betsch, Schoeninger, Murdock-Houwer, Engelking and Isakowitz. The etiology remained unknown; most of the patients were females at the age of menopause.

Hauer's patient, a woman, aged 28, had always been healthy; her menses were normal since her fourteenth year, and she had had normal deliveries at the age of 20 and 21. Since the death of her father, when she was 22 years of age, the flow of tears had ceased. Subsequently emotion would produce no tears, but only congestion of the eyes, headache and heaviness of the lids. Simultaneously, the menses became scant and lasted only one day, instead of three to four as prior to the death of her father, and pregnancy never occurred again. Internal, neurologic and gynecologic examinations revealed no pathologic changes. Finally, the eyes showed constant photophobia, congestion of the palpebral margins and the conjunctiva, papillary hypertrophy of the tarsal conjunctiva, stringy mucous discharge in the lower fornix with xerosis baccilli and epithelial cells of the conjunctiva and cornea. Only two old facets were noticed with the corneal microscope. Schirmer's test continued to show results which were decidedly below the normal value.

Endocrine dysfunction as an etiologic factor is suggested by the cessation of the function of the tear gland and the ovaries. The case is related to those of Betsch and Schoeninger.

K. L. Stoll.

On the Technic of the Extirpation of the Lacrimal Sac. F. W. Weltishteff, Russk. j. opht. **13:** 540 (May-June) 1931.

The author used Duverger's method of additional anesthesia of the sac on fifteen patients with good results. After the usual anesthesia a mixture of 25 cc. of 10 per cent cocaine solution, 75 cc. of 1 : 1,000 solution of epinephrine and 0.5 cc. of methylene blue solution is introduced into the sac two or three times before the operation, every three or four minutes. This method makes the extirpation of the sac considerably easier as complete anesthesia is produced; there is very little or no bleeding, and the contour of the sac is markedly noticeable because of the blue color.

O. Sitchevska.

Lens

A Note on the Inorganic Constituents of Normal and Cataractous Human Crystalline Lenses. G. Mackay, C. P. Stewart and J. D. Robinson, Brit. J. Ophth. **16**:193 (April) 1932.

The problem of methods of preventing the development of cataract in human lenses must be worked out on scientific lines. More precise knowledge of the conditions in which the lens is liable to lose its transparency and the actual changes in the structure and chemical constitution are essential as a base from which to approach this problem.

One must inquire as to what extent substances can be conveyed into the interior of the lens by absorption from the conjunctival sac, the aqueous, the vitreous or through the general circulation. Alternatively, is it possible beneficially to extract undesirable elements through the unbroken capsule of the living lens?

As a first step, it appeared desirable that a fresh series of observations should be made into the chemical condition of cataractous, as compared with transparent, human lenses, and that advantage should be taken of the most up-to-date method of analysis. The following tabulation gives the mean of the analysis of normal and senile mature cataract in per cent:

		Calcium	Potassium	Sodium	Chlorine	Sulphur as SO_4	Phosphorus as PO_4
Normal lens...	Dry wt...	0.029	0.635	0.385	0.097	1.20	0.537
	Ash wt...	1.01	21.8	12.6	3.4	45.8	18.6
Senile mature..	Dry wt...	0.201	0.092	0.622	0.325	1.66	0.386
	Ash wt...	5.49	2.4	27.3	8.5	48.1	8.3

It is to be noted that the various inorganic constituents of the healthy lens are present in concentrations markedly different from those of the same substances in blood plasma, or—which is probably more important—in normal aqueous humor. The lens is thus strongly differentiated from the aqueous humor and from blood plasma. In the cataractous lens, however, this power of differentiation, at any rate from blood plasma, has been partly lost.

There is thus a tendency on the part of the lens to return to, or take on, a composition similar to that of blood plasma, and this tendency is shown in immature as well as mature cataracts. It appears indeed to be progressive and, therefore, to be closely connected with the disease process. A notable exception to this, however, is the calcium, which instead of increasing slightly, as would be expected in a general return to a plasma-like composition, is greatly increased in cataractous lenses, both absolutely and relatively to other ions. There is no evidence that calcium is the first constituent to alter, as one might expect if it were the primary factor in the production of the opacity in the lens.

W. Zentmayer.

Raynaud's Disease and Cataract. D. Cattaneo, Ann. di ottal. **38**: 684 (Dec.) 1931.

The author could find no account in the literature of cataract occurring in association with Raynaud's disease, although Raynaud himself

and others had recognized the contraction of the retinal arteries that accompanies it. His patient was a woman of 47 who noticed poor vision a short time before the occurrence of the characteristic symptoms in the hands and feet, which began three years before she was seen by the author. The right eye showed a mature cataract. The left eye had vision reduced to 6/10 by stellate opacities in the anterior and posterior cortex close to the capsule. These were more dense at the periphery than at the center and resembled apparently the cataracta neurodermatica described by Vogt. General examination revealed an enlarged thyroid and symptoms of hyperthyroidism and ovarian dysfunction. The author reviews the relation of cataract to glandular unbalance, which he thinks played a part in his case. He believes that the direct cause may well have been an intense vascular spasm in the anterior part of the uveal tract, resembling the spasm caused by ergot.

S. R. GIFFORD.

Neurology

NORMAL PUPILLARY REACTION IN PROGRESSIVE PARALYSIS. I. FISCHER, Med. Klin. **28:** 13 (Jan. 1) 1932.

Fischer reports a series of two hundred and three cases of syphilis of the nervous system fourteen of which demonstrated no abnormal pupillary reactions. These cases were seen over a period of two and one-half years, and the pupillary findings were checked by Behr. Eight of the fourteen had other abnormal signs. Among these the foremost were atypical changes in the blood and spinal fluid. The author thinks that further investigations are necessary before any conclusions may be drawn. A chart of the cases with their outstanding conditions and a bibliography accompany the article.

L. L. MAYER.

THE QUESTION OF LOCALIZATION OF THE "SLEEP" CENTER. (APOPLECTIFORM PRESENTATION OF EPIDEMIC ENCEPHALITIS AND OPHTHALMOPLEGIA INTERNA.) E. SCHWAB, München. med. Wchnschr. **79:** 94 (Jan. 15) 1932.

The author reviews the literature on cases reported with lethargy combined with paralysis of both internal and external ocular muscles. He then reports in some detail two cases in which there were apoplectiform seizures, lethargy and paralysis of the ocular muscles. No previous cases are reported in the literature of apoplectiform conditions followed by periods of sleeping due to a hemorrhage. The symptoms suggest the localization of the center for sleep in the posterior portion of the third ventricle and the anterior part of the aquaeductus Sylvii. The outspoken paralysis of the muscles with failure of the upward glance would demonstrate an involvement of the nucleus by pressure on the corpora quadrigemina. The author is unable to explain fully the abducens paralyses, but he thinks that with such a frequent involvement in lesions of the brain one must think of an irritation phenomenon. Two excellent illustrations of the center affected and a bibliography accompany the article.

L. L. MAYER.

Operations

REPOSITION OF THE SEMILUNAR FOLD AS AN OPERATION FOR PTERYGIUM. A. FAZAKAS, Klin. Monatsbl. f. Augenh. **86:** 808 (June) 1931.

The results of many operations for pterygium, especially those of the method of downward transplantation, are frustrated by cicatricial shrinking of the semilunar fold. The author has succeeded in eliminating this contraction by a new method of operating. He makes one incision along the original site of the medial lacrimal furrow, the other along the medial border of the dislocated plica. The conjunctiva between these two incisions is removed. Then the dislodged plica and the pterygium are undermined; one incision is made along the upper and the lower border of the pterygium, extending from the plica to the cornea, and the pterygium is detached from the cornea. The most important step of this operation is the fixation of the plica at its original site by means of from two to three sutures. The pterygium is attached to the bulbar conjunctiva by two sutures at its upper, and two at its lower, border.

K. L. STOLL.

Orbit, Eyeball and Accessory Sinuses

OSTEOMYELITIS OF THE SUPERIOR MAXILLA IN BABIES. D. BOSSE, Arch. f. Ophth. **126:** 465, 1931.

The ophthalmologist should know about this disease, of which the author observed three cases. The babies are usually brought to the ophthalmologist with the symptoms of swelling of one or both lids, sometimes accompanied by signs of an orbital phlegmon. One might first think of a phlegmon starting out from the tear sac which possibility is usually ruled out by the absence of pus in the sac. One might also think of thrombosis of the cavernous sinus. In the latter condition both eyes are affected, and cerebral symptoms are present. The main sign of osteomyelitis of the superior maxilla is a fluctuating abscess in one lid with swelling of the entire maxilla and (usually) pus in the nose. The treatment of these babies should be in the hands of the surgeon or oral surgeon. The abscesses have to be opened; incisions of the lids should be avoided if possible, because they always lead to ectropion and require plastic operations later. Deformities of the superior maxilla are always the result of the osteomyelitis. The parents of the patient should be told that these deformities will become manifest at the age of 6 or later, and that treatment by the oral surgeon will be necessary.

P. C. KRONFELD.

Physiology

THE COMMON LAWS OF EXPERIMENTAL OPHTHALMOTONIC REACTIONS, DIRECT AND CONSENSUAL, PRODUCED BY DIFFERENT PROCEDURES. L. WEEKERS, Arch. d'opht. **48:** 593 (Sept.) 1931.

Weekers discusses the part played by the ocular fluids in the variations, the disorders and the regulations of intra-ocular tension, and also the mode of action of surgical and medical treatment in lowering the tension in glaucoma. In the first part of this paper the various experi-

mental procedures by which it is possible to modify the intra-ocular tension are presented. These include massage, compression of the globe, pricking the ciliary body, subconjunctival injection of irritants, burning of the sclera, contusion of the globe, the injection of a physiologic solution of sodium chloride into the vitreous, etc., and the author's own experiments in which he injected various irritants into the orbit. In the second part of this paper the physiology of the intra-ocular fluid and the part it plays in the regulation of the intra-ocular tension is reviewed. It is as tempting to consider what goes on in the eyes as what happens in vitro in an osmometer, but a large number of facts preclude this. These include the wide variation in the structure of the capillaries in different parts of the eye, those of the ciliary body differing from those of the retina. The very special anatomic character of the canal of Schlemm is pointed out. The permeability of the capillary walls, the rapidity of the circulation of blood and the blood pressure in different vessels are all different in different structures. The author discusses the experimental injection of fluorescein and india ink in relation to the formation and drainage of the intra-ocular fluids. Although Magitot and Duke-Elder consider the intra-ocular fluid a dialysate, they fail to take into consideration the importance of hydrodynamic factors in its formation. Moreover, the laws governing the permeability of a living membrane are far from being fully understood. Agreeing with Magitot that the functions of the intra-ocular fluids are many and important, the author believes that it plays a capital part in the maintenance of intra-ocular tension. The majority of disorders of ophthalmotonus (nontransient, prolonged and persistent), such as are observed clinically, are due to defects in the content of intra-ocular fluids in the globe. This factor is a direct and immediate one, all others, whether local or general, affecting the intra-ocular tension through it. In the third section the author presents evidence to show that variations in the intra-ocular tension are coincident with endocular vasodilatation. He believes that the causes for both hypotony and hypertony must be explained on this basis. It leads to two consequences. 1. The rôle of the aqueous is augmented; more fluid enters and leaves the eye than normally. 2. The relationship between the production and resorption is changed. Compared to normal, the resorption is greater than the production. Hypotony is then due to an overactivity of the resorption of the aqueous.

In order to explain the consensual hypotony in these experiments, he invokes transudates thrown into the blood stream at the time of injury which have a vasodilating activity.

While it has been possible to study the effect of vasodilatation on the intra-ocular tension experimentally, it has so far been impossible to study the effects of vasoconstriction. However, the effects of vasoconstriction are seen clinically, for it is probable that chronic glaucoma is primarily associated with sclerosis of the uveal vessels, especially the capillaries. In chronic glaucoma the circulation of the blood in the eye is defective. The resulting hydrodynamic upset interferes with the resorption of the aqueous and tends to create and maintain a glaucomatous hypertension. The pathologic study of chronic glaucoma deserves reviewing on this basis.

In the last section of this paper, the author presents arguments to support his belief that the favorable effect of all medical and surgical

treatment is due to the production of vasodilatation. In the operative cases, he believes this to be largely the result of traumatism, or possibly of the inflammation of the ciliary body which the operation produces. Medical treatment, he thinks, can be rendered more efficient when intraorbital injections have been more completely investigated.

S. B. MARLOW.

OPHTHALMOTONIC REACTIONS EXPERIMENTALLY PRODUCED BY PRICKING THE CILIARY BODY. L. WEEKERS, Arch. d'opht. **49:** 24 (Jan.) 1932.

Weekers first repeated the experiments of Pesme and Duthil and showed by means of the fluorescein test that the hypotony that follows the use of a round needle without a preliminary rise in tension is probably due to leakage from the anterior chamber. He next presents a series of experiments in which an old cataract knife ground very thin and flat was pushed tangentially through the sclera to prick the ciliary body, making as well sealed a wound as it is possible to do. In these experiments he found a primary rise in tension followed by a rapid fall to a hypotension, which returned slowly to normal. A consensual reaction took place in the opposite eye. The explanation of this phenomenon he has described in a previous paper in which he expressed the belief that vasodilation accounts for it. These general laws apply to all ocular traumatisms, the degree of reaction depending on the severity of the injury. The uninjured eye reacts in a similar fashion but to a lesser degree.

S. B. MARLOW.

THE DIFFUSION OF HEMOGLOBIN IN AQUEOUS, VITREOUS AND BLOOD SERUM. F. P. FISCHER, Arch. f. Augenh. **105:** 431, 1932.

The rate of diffusion of hemoglobin solutions in serum, aqueous, vitreous and a serum ultrafiltrate was measured. It was found that hemoglobin diffuses much quicker in serum than in any other solutions of colloidal or molecular dispersion and at the same time shows a considerable variation and abnormal character of diffusion. The acceleration of the diffusion in serum is due to a change in the particulate size and an increased imbibition of the hemoglobin micellae. This is due to the peculiar properties of the serum proteins. These experiments attempt to explain the imbibition of blood pigments by the cornea, lens and vitreous in cases of blood staining.

F. H. ADLER.

Refraction and Accommodation

THE REFRACTION OF CASES OF CONGENITAL TORTICOLLIS ASSOCIATED WITH HEMIATROPHY OF THE FACE. A. SORSBY and M. SHAW, Brit. J. Ophth. **16:** 222 (April) 1932.

Nine cases of congenital torticollis associated with definite hemiatrophy of the face were observed. Not only was the affected side of the face grossly smaller than the other, but the diameters of the affected orbit were also smaller. In only one case was there marked difference in the refraction of the two eyes, so that it is justifiable to conclude that influences in the development of the orbit do not affect the state of the refraction of the eye.

W. ZENTMAYER.

THE PROBLEM OF ACCOMMODATION AND CHANGES OF REFRACTION. (REMARKS CONCERNING THE WORK OF DR. HANS CASPARY). R. GUTZEIT, Med. Klin. **28:** 119 (Jan. 22) 1932.

Caspary's work in a measure gives approbation to the ideas of Bates and his school. He reinforces strongly the idea that the eye is adapted for near work by the contraction of the ciliary muscle, thus strengthening the curvature of the anterior lens surface, and also that the extra-ocular muscles play an important rôle in changes of refraction, because the eyeball is not a fixed body. Caspary believes that through the tension of the superior, inferior and internal recti muscles when the eye converges the axis of the eye is lengthened, aided by the fanning of other musculature. And, finally, as the tension of the contracting ocular muscles is increased, a shortening of the curvature of the cornea occurs.

According to Caspary, three forms of accommodation are set in order: (1) the previously recorded, scientifically based accommodation of the lens by means of the ciliary muscle, according to Helmholz; (2) the outer or axis accommodation, caused by the extra-ocular muscles; also called photographic accommodation, as the picture screen (the retina) is pushed off from the optical system; (3) the corneal accommodation by shortening of its measure of curvature following the increased tension of the eyeball caused by the contraction of the extra-ocular muscles.

The author believes that the following facts speak against the actual rarity of outer or corneal accommodation, viz.: the entire elimination of accommodation with atropinization although convergence remains; the increased tension of accommodation with physostigmine (eserine), also without convergence, and the isolated accommodation paralysis by damage to the innervation of the ciliary musculature (belladonna and meat poisoning, diphtheria, syphilis). The absence of accommodation in aphakic eyes after cataract operation fails to reveal other than lenticular accommodation. Accommodation and convergence of the lens are so firmly blended that it is difficult to imagine any other purely mechanical mechanism, which also speaks against Caspary's hypothesis. In convergence the medialis contracts and the lateralis relaxes; the former is pulled away from the globe and the latter lies in a discrete mass on the globe; surely, the superior and inferior muscles, which play so small a part in convergence, can have little effect in pulling back the globe and thus causing a lengthening of the axis of the eyeball.

In other ways the author shows the unscientific and rather psychic effect of the Bates method and emphasizes his belief that it should be condemned.

L. L. MAYER.

Retina and Optic Nerve

HEMATOMA OF OPTIC NERVE SHEATH. ANONIO J. MANES, Arch. de oftal. de Buenos Aires **6:** 575 (Oct.) 1931.

Two cases of hematoma of the optic nerve are reported. In one hemorrhage from the optic nerve vessels followed a gunshot wound. The ocular symptoms consisted of exophthalmus from retrobulbar hemorrhage, ptosis, opaque vitreous from hemorrhagic detachment of the retina with complete blindness with preservation of the pupillary reflex in one eye and moderate optic neuritis of the other. Decompression of cranial cavity and orbit was followed by improvement of

some of the local conditions; the sight of the affected eye was, however, permanently lost. The second case was one of fracture of the base of the skull, involving the petrous portion of the temporal bone, in which the clinical history was typical, supplemented by a sixth nerve paralysis.

The reports are supplemented by general considerations on the symptomatology of hematoma of the optic nerve sheath.

C. E. FINLAY.

TWO CASES OF SPASM OF THE CENTRAL ARTERY OF THE RETINA. PAULINA SATANOWSKY, Arch. de oftal. de Buenos Aires **6:** 617 (Nov.) 1931.

One case of spasm of the central retinal artery was observed in the course of a malignant nephrosclerosis, the observer having the occasion to note a let up of the spasm during an ophthalmoscopic examination, with a consequent improvement in sight. The opthalmoscopic picture during the spasm consisted in one of retinal ischemia with slight retinal edema, with disappearance of the arteries, the pupils being myotic and not reacting. The second case occurred in the course of postneuritic atrophy of syphilitic origin in one eye, the arteries of which were filiform, a dilatation being observed during an ophthalmoscopic examination together with a slight visual improvement.

In commenting on these cases, the author draws inferences as to the pathogenesis of the amaurosis occurring in toxemia from ethylhydrocupreine hydrochloride and in that following profuse extra-ocular hemorrhages, which he considers of a similar nature.

C. E. FINLAY.

OBSERVATIONS ON GONIN'S OPERATION. AMSLER, Arch. de oftal. hispano-am. **31:** 664 (Dec.) 1931.

After referring to de Wecker and Leber's pathogenic theories resuscitated by Gonin, the author includes under the name of Gonin's operation all the operative measures for obliteration of retinal ruptures by thermopuncture. He insists that there must be a retinal rupture before the occurrence of an idiopathic detachment.

The most important point in connection with the operation is the correct localization of the rupture, which implies an attentive, persevering and observing eye behind the ophthalmoscope.

Gonin attributes the production of the retinal rupture to a histopathologic lesion in the vitreous, without absolutely denying the influence of a latent inflammation of the choroid, the abnormal adhesions dragging on the retina and producing its rupture.

In connection with the necessary precise localization of the site of the retinal rupture, the author refers to the simple and precise methods employed at Lausanne, which largely depend on the personal equation, to avoid which Guist, Fischer and Weve have constructed apparatuses for the certain and correct localization of the ruptures. The author refers also to Arruga's localizing hook, which allows an ophthalmoscopic confirmation during the operation.

When an obliteration of the rupture is sought by means of a more or less extensive deep reaction, the localization of the site of rupture

need be only approximate. The chemical cauterization of the surface by the method of Guist may be made in the region of the rupture, the objective being a fixation of the retina to the ocular wall and destruction of the pathologic choroidal focus. The author next discusses the objections offered of possible secondary adhesions producing future ruptures. These fears are not upheld by facts. Lindner and Guist's chemical cauterization of the surface seems to show that it may be possible to obtain an obliteration of the rupture without loss of vitreous.

As regards operative results, it is first necessary to determine what is considered as an "improvement" and what a "cure." The criterion may be subjective or functional or objective or anatomic. As regards the former, only central vision is considered by the patient, but this is fallacious and temporary; the author considers the perimetric examination as more reliable. The best statistics are those of Gonin, which show 53 per cent of successes; Arruga's and the Moorfield's (32 per cent), come next in order. The author has operated in forty cases of detachment with twenty readhesions and four improvements.

The indications and contraindications are always difficult to determine.

Under the caption of "idiopathic," the author includes all nontraumatic cases and those complicating ocular diseases such as retinitis albuminurica.

C. E. FINLAY.

RETINITIS PROLIFERANS AND MULTIPLE ANEURYSMS. HORACIO FERRER, Rev. cubana de oto-neuro-oftal. **1:** 1 (Jan.-Feb.) 1932.

This is a report of a case, illustrated with a colored plate, of retinitis proliferans, in which, apart from usual lesions, some fifteen minute vessels sprang from the cribriform plate and project fan-shaped into the vitreous, being held together by an imperfect membrane, and after anastomizing with one another, terminate in about thirty-five round bulbs, which the author considers aneurysms. The arterial nature of these vessels is shown by their disappearance or thinning on pressure applied to the eyeball and on their refilling when this was released.

C. E. FINLAY.

ANGIOMATOSIS RETINAE AND RETINITIS EXSUDATIVA COATS. E. VON HIPPEL, Arch. f. Ophth. **127:** 27, 1931.

The author gives the pathologic findings in eight eyes sent to him with the clinical diagnosis of glioma or suspected glioma. The first patient was a boy, 4½ years of age; only a retinal detachment had been seen *intra vitam*. The sections showed a definite angioblastic tumor in the retina with extensive cyst formation, otherwise the picture of an exudative retinitis which is always present in late stages of angiomatosis retinae. The main difference between the latter and the various pictures which are comprised under the name Coat's retinitis is the characteristic fundus picture in angiomatosis (which, however, is not often actually seen in children because of the technical difficulties) and Lindau's complex general symptoms (angiomas of the cerebellum, cysts in the pancreas, etc.) which never occurs in Coat's disease. The anatomic differentiation of the two diseases is almost impossible because vascular malformations are the most important feature in both conditions. The second and third case showed the anatomic "characteristics" of Coat's

disease, in the second case, apparently produced by a congenitally syphilitic vascular process, and in the third case, produced by a vascular malformation. The fourth case was of a tuberculous nature, the fifth, apparently, had a peculiarly localized metastatic ophthalmia. Case 6 showed an abscess in the vitreous cavity; case 7 a very severe caseous tuberculosis, and case 8 an unusually severe form of congenital syphilis.

P. C. KRONFELD.

THE INFLUENCE OF MALARIA ON THE COURSE OF RETROBULBAR NEURITIS OF SYPHILITIC ETIOLOGY. L. B. KATSNELSON, Russk. j. opht. **14:** 229 (Sept.) 1931.

The author reviews the literature on treatment of all forms of general syphilis and syphilitic conditions of the eye by malarial vaccines.

He reports a case of retrobulbar neuritis that progressed to optic atrophy, was resistant to syphilitic and nonsyphilitic treatment and improved spontaneously after a few malarial attacks. A woman, aged 30, was under the author's care since 1925, when she first consulted him with acute paralysis of the sixth nerve. Although the Wassermann reaction was negative, a course of antisyphilitic treatment was given which resulted in complete recovery. Two years later her right eye was affected with retrobulbar neuritis, and the following year the left eye, too, was affected, the vision failing rapidly in that eye. There was a central relative scotoma for white and an absolute scotoma for colors in the right eye, and an absolute scotoma for white with no color perception in the left eye

All search for a focus of infection was negative. The spinal fluid, examined twice after two new courses of antisyphilitic treatment, was normal. Tuberculin therapy was applied without any results. In 1930, the patient took ill with malaria, and Plasmodium vivax was found in her blood. After the fourth attack the patient noticed that she could see clear with the right eye The vision of the right eye was improved from 0.3 to 0.9, that of the left from 0.06 to 0.09. The central scotoma disappeared, and the optic nerve was of a pink color in the right. The visual fields and fundi were unchanged in the left eye.

The author believes that the therapeutic experiment done by nature itself is worth while drawing the attention of the ophthalmologists in trying malarotherapy in severe neurosyphilitic conditions of the eye.

O. SITCHEVSKA.

Tumors

EPITHELIOMA OF THE LIMBUS. TERRIEN and COUSIN, Arch. d'opht. **48:** 622 (Sept.) 1931.

The authors report briefly the case of a man with an epithelioma of the limbus, which was excised first in June, 1929. It recurred in October, 1930, and was unsuccessfully treated by galvanocauterization but was finally controlled by diathermic coagulation. They had been on the point of advising enucleation when they began using the high frequency current with apparent success. The authors point out the great differences of opinion that are to be found in the literature on tumors of the limbus, including epitheliomas. They discuss this form of tumor of the limbus from the standpoint of age incidence, site of origin and

development. The statement is made that the constant presence of pigment in the region of the limbus is probably the main reason why epibulbar tumors have a predilection for this area. While some of them may arise from congenital nevi, the authors feel that Veil is a little too positive in his belief that the majority of limbal epibulbar tumors are epithelial tumors arising from nevi. Its invasion of the orbit and the lid is most to be feared. Extension to deeper structures is rarely observed, and involvement of the lymph glands occurs only in the terminal stages. Its marked tendency to recur constitutes its chief danger to the patient. The retention of good vision presents a serious obstacle to enucleation, which the patient will consent to only as a last resort. Treatment of cases of this kind has been the subject of much controversy. Surgical excision with cauterization of the base seems in the past to have been most satisfactory, but not entirely free from the danger of recurrence. Radium treatment has met with varying success. The authors' experience leads them to suggest the use of the electric knife followed by the application of high frequency current at the base of the tumor as the best treatment.

S. B. MARLOW.

TUMORS OF THE EYES WITH PARTICULAR CONSIDERATION OF THERAPY. J. BÖCK, Wien. klin. Wchnschr. **44:** 1199 (Sept. 18) 1931.

After a short comment on the group of benign diseases of the lid, their therapeutic procedures are reviewed. Every one knows of the good results of radium emanations on cavernous angioma of the lid and conjunctiva. For nevi, Bucky's Grenz rays or carbon dioxide snow often gives good results. For other benign conditions one may recommend extirpation, as the majority are not impressed with radiation therapy. A warning must be given before radiation treatment of pigmented and unpigmented nevi of the lids especially, and also the conjunctiva, that these may go on to a malignant state. In carcinoma of the lid, surgery is preferred, as a more radical removal may be done, with later radiation therapy.

In far advanced carcinoma with an extensive defect of the lid, which can be removed only by sacrificing the eye, the tumor mass should be excised as wide as possible, preserving the eyeball if possible, and the remaining tumorous tissue should be treated with radium emanations. A well known diagnostic obstacle is seen in carcinoma of the glands of the lid, which may frequently be confused with chalazion. A protection against a false diagnosis is given by an histologic examination in each case in which the condition is suspected. Radical exenteration and radium radiations have rendered the most efficient therapy up to the present. Regarding intra-ocular tumors, the progress in therapy has changed little in the last years. With surer diagnoses and forsight, enucleation of the eye will occur oftener. In sarcoma of the uvea and glioma of the retina, radiation has given little of real worth. In case of a unilateral glioma, irradiation has been impractical up to the present.

H. Wintersteiner has found glioma of the retina in both eyes in about 19 per cent of the cases. In these cases the eye with the more advanced condition is enucleated, and the other eye treated with the roentgen rays, as there are a few cases reported in the literature in which the growth has been sucessfully retarded with the roentgen rays. As a rule this does not occur, and thus in the unilateral glioma enuclea-

tion is resorted to. In sarcoma of the uvea, no results have been obtained up to the present with irradiation. Fuch's statistics seems to prevail; only 6 per cent are permanently healed. It is noteworthy that in sarcoma of the uvea metastasis takes place in far distant organs (liver) and usually after many years. The most difficult for diagnosis are the tumors of the orbit. In these cases roentgenograms of the skull, together with general examination, will help in the diagnosis. An exploratory surgical exposure of the orbit is indicated in doubtful cases. In this way benign tumors like endothelioma of the optic nerve sheath are discovered. If a malignant condition is found total exenteration of the orbit, with radiation following, is indicated.

L. L. MAYER.

Uvea

ANGIOMA OF THE CHOROID. E. VON HIPPEL, Arch. f. Ophth. **127:** 46, 1931.

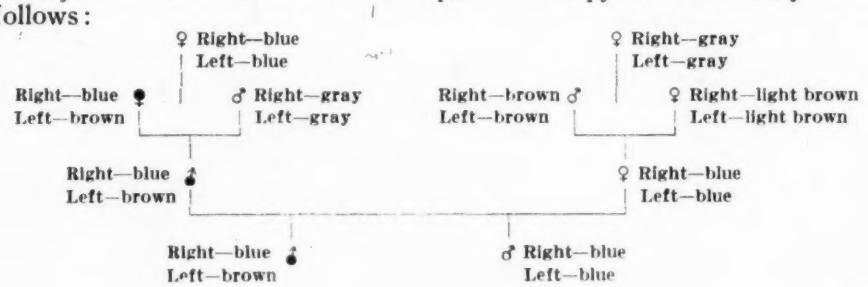
The author describes two cases of angioma of the choroid. The first case permitted ophthalmoscopic (one beautiful colored drawing) and anatomic examinations, the second case only anatomic examination. In the first case the tumor was located below the disk, occupying an area of 4 by 3 disk diameters. The macula was slightly opaque. The anatomic basis of this haziness was a thin layer of tissue which looked like connective tissue, but could be derived from the pigmented epithelium. These formations outside of the area of the tumor seem quite frequent in cases of angiomas of the choroid. Von Hippel pleads for their strictly ectodermal origin, and, therefore, calls it a tissue resembling connective tissue.

As to the ophthalmoscopic differential diagnosis, von Hippel formulates his opinion as follows: The angioma presents itself like any other tumor occurring in the fundus (sarcoma, tuberculoma). Its color and other features may suggest its being an angioma, but most of the time these qualities are too vague to permit a definite diagnosis. More certain symptoms are angiomas found in other parts of the body, isolated dilation of one vorticose vein or general dilation of the choroidal vessels in one area of the fundus.

P. C. KRONFELD.

A FAMILY TREE OF HEREDITARY HETEROCHROMIA OF THE IRIS. H. NOWAK, Deutsche med. Wchnschr. **58:** 94 (Jan. 15) 1932.

The rarity of heterochromia of the iris and a fairly complete family history are the reasons for the report. A copy of the family tree follows:



Three generations are shown that have a blue right iris and a brown left iris. The hereditary nature of the condition is thus demonstrated.

L. L. MAYER.

TUBERCULOUS ETIOLOGY OF CHRONIC UVEITIS AND OTHER INFLAMMATORY DISEASES OF THE EYES. J. MELLER, Wien. klin. Wchnschr. **45:** 33 (Jan. 8) 1932.

The article is a résumé of the author's views and presents reasons for his views on the question. The rarity of the diseases discussed, the inability of the internist to recognize potential tuberculous lesions, the difficulty of finding the organism and the complications caused by other bodily diseases make the solution of the problem difficult, in spite of the fact that such lesions are recognized clinically by the experienced as of tuberculous origin. Positive blood cultures found by several authorities, together with typical pathologic changes as found in positive tuberculous lesions, have aided the clinician in substantiating his beliefs. Even sympathetic ophthalmia may some day be shown to have an underlying tuberculous basis, as the pathologic findings are so similar. The author seems convinced that the basis for chronic uveitis is tuberculosis.

L. L. MAYER.

Therapeutics

THE USES AND MISUSES OF VACCINES. J. A. BRAXTON HICKS, Brit. M. J. **2:** 650 (Oct. 10) 1931.

The writer says that generally speaking, vaccines cannot be used in cases of great urgency; antiserums are of value only under those conditions. Vaccine therapy is of the greatest use in prophylaxis, in which case, of course, stock vaccines must be used. Detoxicated vaccines are generally useless as specific remedies. The cases in which vaccines will be successful occur more in young than in old persons. Vaccines should never be given in conditions in which there is a high temperature. As a rule, the author prefers to give small doses, at frequent intervals, gradually lengthening the intervals and increasing the doses as tolerance increases. The most important reaction is the local induration.

In tuberculous lesions, the author prefers old tuberculin, and he has obtained particularly good results in certain eye and skin conditions, in early tuberculous joints, but particularly in tuberculous glands.

In chronic stages of gonorrheal prostatitis, he has not had much success with stock vaccines, but he thinks that a stock vaccine plus the organisms of secondary infections, which cause many of the late manifestations obtained after massage, are often helpful.

He also is a believer in the prophylactic use of vaccines against colds.

A. KNAPP.

REFLECTIONS CONCERNING THE EMPLOYMENT OF HIGH FREQUENCY IN OPHTHALMOLOGY. M. DE TREIGNY, Ann. d'ocul. **168:** 972, 1931.

High frequency in its different forms is a method that is perfectly applicable to ocular diseases, but requires prudent application as regards indications and cases. The principal indications are in the following

conditions: tumors of the cutaneous glands, xanthelasma, etc., epilation of the eyelashes and for the treatment of cicatrices. It is an established fact that in these conditions, at least, good results have been obtained.

S. HANFORD MCKEE.

TREATMENT OF HYPOPYON ULCER BY PARACENTESIS WITH THE GALVANOCAUTERY. G. PACALIN, Arch. d'opht. **48:** 498 (July) 1931.

Pacalin believes that paracentesis of the cornea is one of the most valuable methods of treatment for ulcers of the cornea. He prefers puncture of the cornea with a fine cautery point because of its simple performance without the necessity of fixation and because it can be repeated several times. Moreover, a cautery puncture does not open up avenues of infection as does the use of a knife, and it is therefore sounder in principle in an infected field. He emphasizes the necessity of having a fine cautery point which must be plunged into the anterior chamber and withdrawn quickly to prevent too much corneal scar. The selection of the area to puncture depends somewhat on the location of the ulcer. If the ulcer is peripherally situated, the puncture is made through it near its highest margin. If centrally located, the puncture may be made through clear cornea about half way between the limbus and the center of the cornea. The best results are obtained when the aqueous escapes in a jet and when the puncture is made high enough so that the hypopyon does not entirely escape but spreads out over the surface of the iris.

S. B. MARLOW.

NEUROMEDICAL OBSERVATIONS CONCERNING THE TREATMENT OF TABETIC OPTIC ATROPHY. E. WEINBERG, Deutsche med. Wchnschr. **58:** 173 (Jan. 29) 1932.

After reviewing the literature, the author adds his own experiences and draws the following conclusions concerning the treatment of tabetic optic atrophy: 1. The optic atrophy in tabes is usually an early symptom and generally appears first in the treatment of the nervous system, when the process shows progressive tendencies. 2. The specific-nonspecific treatment with arsphenamine and bismuth preparations on the one hand, and with the so-called "fever cures" on the other, has been regarded as the method of choice in tabes. It has been used also in progressive cases complicated by optic atrophy. 3. On the basis of the neurologic material seen by the author, there is little hope that optic atrophy will be cured. A bibliography accompanies the article.

L. L. MAYER.

CAN BISMUTH ALONE BE SUFFICIENT IN THE TREATMENT OF SYPHILIS OF THE EYE? I. I. KASAS, Russk. arch. opht. **8:** 267 (March-April) 1931.

The author has been using bismuth therapy for syphilitic infections of the eye in his clinic since 1924. During the first year he observed many complications, such as iritis, scleritis and exacerbations of optic atrophies. For the past six years he has been using bismuth at the

end of mixed treatment in the following order: For two weeks injections of mercury and iodides are used; on the third week neoarsphenamine is added; after this course is finished, injections of bismuth are employed. No complications have been observed since this mode of treatment was adopted.

The author believes that Spirochaetae lose their aggressiveness under the influence of mercury, iodides and neoarsphenamine, and that the patient thus can be saved from the provocative action of bismuth, or from the biotropic reaction. His advice, therefore, is never to use bismuth alone, nor at the beginning of the antisyphilitic treatment unless the other three drugs are applied first.

O. SITCHEVSKA.

Toxic Amblyopia

RETROBULBAR NEURITIS DUE TO THALLIUM POISONING FROM DEPILATORY CREAM. W. MAHONEY, J. A. M. A. **98:** 619 (Feb. 20) 1932.

The following is a summary of the symptoms manifested by the patients:

CASE 1.—A woman, aged 29, had peripheral neuritis, falling hair, failing vision, central scotoma, retrobulbar neuritis and nervousness.

CASE 2.—A woman, aged 28, had peripheral neuritis, failing vision, central scotoma, retrobulbar neuritis and loss of hair.

CASE 3.—A woman, aged 35, had peripheral neuritis, nervousness, falling hair, low visual acuity with central scotoma, retrobulbar neuritis, bilateral motor and sensory involvement of the legs.

These patients entered a neurosurgical clinic as "intracranial tumor suspects." There was in all a history of long continued use of a preparation containing a liberal percentage of thallium. The failing vision had been checked and improved by discontinuing the employment of what, therefore, is the most probable source of the disorder.

W. ZENTMAYER.

OCULAR PLUMBISM IN CHILDREN. J. L. GIBSON, Brit. J. Ophth. **15:** 637 (Nov.) 1931.

The reason for the publication of this and previous papers by the author is a desire to remove from young Australian children the danger of ocular plumbism between the ages of 2 and 8, and also to make isolated cases in other places and other countries less likely not to be diagnosed as due to lead poisoning—at least to bring lead into the differential diagnosis of such cases in young children.

The author summarizes the clinical findings in the cases dealt with in the paper as follows: There is no pyrexia. The children with mild cases are brought by their mothers because a recent internal squint has developed. The squint is due to paralysis of one or both external recti. Papilledema up to 6 diopters is found, due to increase of intracranial tension. The severe cases are more evidently cases of lead encephalopathy. Explanation is given of why soluble lead is available, and why and how it is ingested. Lead is found in the urine. No albumin is found in the urine.

W. ZENTMAYER.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

ALGERNON B. REESE, M.D., *Secretary*

Jan. 18, 1932

MARK J. SCHOENBERG, M.D., *Chairman*

PROJECTION OF MICROSCOPIC SPECIMENS ILLUSTRATING HOLE IN THE MACULA, LYMPH FOLLICLES IN THE CILIARY BODY AND "POSTERIOR VORTEX VEINS." DR. LOUISE H. MEEKER.

Hole in the Macula.—CASE 1.—A woman, aged 38, with diabetes, shows myopia and bilateral congenital perinuclear cataract. Iridectomy, needling and trephinement were performed within one year. The eye has been blind for nine months. The hole in the macula is one-half disk diameter and is associated with flat detachment and general edema of the retina. There are also small retinal hemorrhages.

CASE 2.—The patient is a man, aged 66. The long diameter is 26 mm. The eye has been blind for six months; the onset was gradual. The urine contained 3.3 per cent sugar. Detachment of the retina was recognized clinically. The hole in the macula is one-third disk diameter and extends through all the layers. The detachment of the retina is recent, and there is little associated retinal edema. There are associated congenital anomalies, such as nevi of the iris, iris processes and small temporal conus.

True Lymph Follicles in the Ciliary Body.—A boy, aged 15 years, has phthisis bulbi. The eye has been blind since infancy. The Wassermann test was negative. There are several deep opacities on the cornea, and seclusio and occlusio pupillae. Râles are evident in the lungs. The general choroiditis suggests tuberculosis with diffuse epithelioid cell proliferation and associated giant cells of the Langhans type. The retina is entirely disintegrated, and the lens has been absorbed. The long posterior ciliary nerve loops outward under the conjunctiva.

Posterior "Vortex Veins" (One Without Associated Conus).—CASE 1.—A woman, aged 58, has glaucoma. The long diameter is 28 mm. The posterior vortex vein is without associated conus, but there are other congenital anomalies, such as looping outward under the conjunctiva by both the long posterior ciliary nerve and the artery. Trephinement was performed. The patient has had cortical cataract and later iridocyclitis plastica. Schlemm's canal is distended with blood. The sinus angle is obliterated. There are occlusio and seclusio pupillae. The excavation of the disk is filled by connective tissue. Dense collections of lymphocytes surround the posterior "vortex veins."

CASE 2.—The patient is a man, aged 44 years. The long diameter is 27 mm. There is traumatic iridocyclitis. One eye is blind. The other eye is myopic, with a posterior vortex vein and temporal conus. The temporal conus exceeds the disk diameter in width. The eye has always been weak.

DISCUSSION

DR. BERNARD SAMUELS: Hole in the macula is seen rather rarely clinically, but under the microscope it is seen more frequently, because it occurs in many conditions in which the eye is so damaged that one is not able to observe the fundus clinically. It occurs in tumors of the orbit that make pressure on the globe. It is especially apt to occur after foreign bodies in the globe, and also after extractions of cataracts in which a great deal of lens matter has been left. It is really not a hole, but a round cystic space, as a rule. Clinically, it resembles a hole because the margins are very sharp, which gives a disklike effect.

Concerning the glandular structures in the ciliary body, there are great variations in the reticulum of Heinrich Müller. Every once in a while one sees an evagination of the epithelium which extends far into the vascular layer of the ciliary body. Such evaginations can have scarcely anything to do with the secretion of the aqueous.

Regarding the posterior ciliary veins, with the ophthalmoscope they are commonly observed in myopic eyes in which there has been atrophy of the choroid. I have no doubt that they occur much more frequently if one could see them, but they are obscured by the stroma of the choroid. They might be found oftener under the microscope if one looked for them.

DR. MARTIN COHEN: If serial sections had been made, might they not have shown some disturbance in the nutrition of the macular area which could explain the hole in the macula in the case of diabetes?

DR. LOUISE MEEKER: I think that the hole in the macula in the case of diabetes is part of the general edema seen in the detached retina. The detached retina with subretinal exudate is evidently due to the generalized arteriosclerosis of the choroid in this eye.

MUSCLE CASES. DR. RUDOLF AEBLI.

Five cases are shown from the muscle clinic of Dr. James W. White. Three of these patients have congenital paralysis of both elevator muscles in one eye.

CASE 1.—A boy, aged 4 years, has paralysis of the left inferior oblique and of the left superior rectus muscles, and in the primary position has complete ptosis of the left lid. With the eyes down and to the left, and down and to the right, he fixes with both eyes and fuses with the stereoscope. When the right eye is covered, the left remains wide open. The pseudoptosis in this case is due to the diplopia in the primary position and in the upper fields.

CASE 2.—This patient also has congenital paralysis of both elevators of the left eye.

CASE 3.—A boy, aged 9 years, had paralysis of both elevators of the right eye. In the lower field he had binocular single vision, and in the upper fields marked vertical diplopia. As a result, he held his chin up

and tilted his head sharply backward. An advancement of the right inferior oblique and of the right superior rectus was performed. The area of single vision was thus raised to include the primary position, and in consequence, the head is held erect and straight.

CASE 4.—A case of traumatic origin shows paralysis of the right inferior and the right superior rectus. The other branches of the third nerve are normal. I believe this to be due to a peripheral hemorrhage.

CASE 5.—The patient had double hypertropia with severe alternating esotropia. The hypertropia was present in all fields, somewhat greater in the upper fields, and was due to a bilateral paralysis of the superior rectus, with an overshoot of the opposite inferior obliques, and a secondary contracture of the direct antagonists, the inferior recti. The vertical element was first corrected by tenotomy of both inferior oblique muscles. The child was then given stereoscopic fusion exercises with the help of a 15 diopter prism, base out, in each eye. Six months later, a bilateral recession of both internal recti was done. The child has an excellent result and fuses with the stereoscope. The refractive error is negative, and vision is 20/20 in each eye.

DISCUSSION

DR. JAMES WATSON WHITE: Congenital paralysis of both elevators of one eye has been observed in six patients during the past year. Only one came to operation, and the result was not satisfactory. In the boy shown by Dr. Aebli, much practical benefit resulted. He was operated on about four years ago.

In cases of either convergent or divergent strabismus, combined with vertical strabismus, it is often a question on which condition to operate first. My choice is determined by correcting both deviations with the cover test. If the correction of the vertical deviation has no effect on the amount of convergence or divergence, the lateral deviation is operated on first. If the vertical deviation is noncomitant or above 10 degrees in the primary position, I attack the vertical deviation first. If the lateral deviation is decreased when the vertical deviation is corrected by prisms, the vertical deviation should nearly always be corrected surgically first with the understanding that the lateral deviation may have to be operated on also. This can often be told definitely by the effect when the vertical elevation is corrected by prisms.

In operating, it is often possible to correct both deviations at one time, if the patient is under a local anesthetic, the vertical deviation usually being corrected first. In a patient under a general anesthetic, it is safer to correct one deviation first and to await the result of this intervention before performing the second. This may be done before the patient leaves the hospital, in cases in which correction of the vertical does not affect the lateral, but when improvement is probable it is better to postpone the second operation. This produces better final results and decidedly improves the operator's judgment in dealing with complex cases.

DR. ARTHUR A. GALLO: In the first case just demonstrated, rather than paralysis of the superior rectus and inferior oblique of the left eye, I think the patient has ptosis of the eyelid, of the pseudoparalytic variety, due to spasticity of the orbicularis rather than to paralysis of the levator palpebralis.

Another sign related to the ptosis and nicely illustrated by this case is the so-called "phenomenon of Marcus Gunn," which consists in disappearance of the ptosis the moment the patient opens his mouth widely, and in alternating movements of opening and closing of the eyelid corresponding to the opening and closing of the mouth during mastication.

DR. MARK J. SCHOENBERG: I should like to ask Dr. Aebli what is his technic for making an advancement of the inferior oblique.

DR. RUDOLF AEBLI: I did not state that there was paralysis of the levator, and the fact that when the right eye is covered the boy opens the left eye shows that no such paralysis is present. I do not believe that the ptosis is due to spasm of the orbicularis, because when the right eye is covered the left eye is constantly open. There is a loss of elevating action of both the left superior rectus and the left inferior oblique. In looking down and to the left, and down and to the right, this child fixes with both eyes, and when tested with the stereoscope he was able to fuse in the lower left and the lower right fields; but as soon as the eyes move up into the primary position, or above, double vision develops, and I think he closes the left lid to blot out the double vision. I think that the condition is a congenital abnormality of the muscles or nerves.

In reply to Dr. Schoenberg, I have had little experience in cases of advancement of the inferior oblique. In the two cases in which my associates and I operated, we made the usual incision parallel to the infra-orbital rim, caught the inferior oblique with the hook and sewed it to the periosteum just inside the rim of the orbit; in this way we secured a greater pull from the inferior oblique.

THE STUDY OF HUMAN CONSTITUTION FOR THE OCULIST. DR. GEORGE DRAPER.

It is said that the eye is the organ of vision. Yet it is common knowledge that when several persons, possessing equally perfect eyes in an optic sense, observe the same object, each will perceive it a little differently. Such discrepancy of perception can be explained only by the special or peculiar reactivity of each person to the ocular stimulus. It is not the eye in the sense of an optic instrument that determines one's reactions to the effects of its function, but the pattern of the personality behind the eye. This principle is likewise true of all the other organs of sense. Consequently, it may be held that so far as one's reaction to environment is concerned, the eye is but one of an aggregate of five parts which together form a vast composite organ of sensation.

Methods for studying the total personality are now available, and have been carried out in great detail. It is not possible at this time to describe them fully, but the methods that have been devised for the study of the individual subject are all published, and are of a nature that does not prevent their being used by every practitioner after a brief period of training. They should merely be looked on as part of the equipment for general physical and psychologic examination.

The influence of the endocrine glands on the sympathetic nervous system is so important in determining what might be called the flavor of the personality that they must be investigated. Probably there is no condition in the eye that has interested oculists more than glaucoma. This condition has long been supposed to have relationship to endocrine

imbalance, and to changes in the tonus and excitability of the parasympathetic and sympathetic phases of the nervous system. Such observers as Laqueur, Favory and Stenico have written extensively on this point. But there is little definite knowledge as yet. The situation seems to rest at the moment with the implication that glaucoma is related to glandular imbalance and to disturbance of the normal tonus of the sympathetic nervous system. This state of affairs naturally incites one to further study of the patients who have this disease.

TUMOR OF THE PITUITARY BODY WITH TERMINAL MENINGITIS. DR. JOHN A. BILLINGSLEY.

A 48 year old colored man who was able to carry on his work as an insurance agent, first noticed visual disturbance only eight days before death; he described this as a "gloominess" of his left eye. The condition of the left optic nerve, as observed at autopsy, showed that he had been using only his right eye, and vision in this eye was apparently cut off on the morning of the acute attack by sudden pressure of the tumor on the right optic nerve, due probably to hemorrhage or edema within the tumor. The accompanying acute meningitis, diagnosed by a neurologist from the spinal fluid cell count, was the immediate cause of death, and the microscopic observation of an inflammatory exudate at the base of the tumor and the thinned-out, eroded wall of the sella with the clouded sphenoids gives a probable clue to its etiology.

TREATMENT OF TOXIC AMBLYOPIA WITH VASODILATORS. DR. W. F. DUGGAN.

Toxic amblyopia usually requires from two months to a year for a complete cure. Also, in many cases, there is a complicating factor which seems to delay or prevent recovery. In 1930, Pflimlin reported fifteen cases of toxic amblyopia in which the patients had been treated with a vasodilating agent, essentially a solution of sodium nitrate in an isotonic salt solution, for intravenous use. The average time of treatment required for cure was 5.2 weeks, and the average number of injections was 12. Of the thirty eyes involved, 23 attained vision of 20/50 or better, 19 attained 20/40 or better, and 15 attained 20/30 or better. One of his patients improved under treatment with the compound sodium nitrite solution despite the continued use of alcohol and tobacco.

At the Herman Knapp Memorial Eye Hospital my associates and I have treated six patients with toxic amblyopia with a compound sodium nitrite solution, intravenous injections being given every day. On the first three days, 40 mg. of the sodium nitrite solution was given. For the remaining injections, 100 mg. ampules were used. The patients received from six to fifteen injections. Of the twelve eyes treated, eight attained vision of 20/30 or better and two 20/50, and in the other two the condition was essentially unchanged. In four of the cases, the maximum improvement occurred in from three to fourteen days after treatment was begun, and one patient (who received the injections every other day) required five weeks to attain vision of 20/30 and 20/25 in the two eyes. In the five cases in which improvement occurred, the fields showed a correspondingly rapid improvement.

I feel that the use of a vasodilator will shorten the time required for the return of serviceable vision in toxic amblyopia, and that eyes that come to a standstill or do not improve with the usual treatment deserve a trial with a vasodilator.

DISCUSSION

DR. ARNOLD KNAPP: In cases of toxic amblyopia in which the patients do not recover vision, although they have apparently stopped using alcohol and tobacco, one has been led to suppose that there is still some active factor present. In these cases, as Dr. Duggan has reported, definite improvement resulted from the use of a compound nitrite solution, and I think that this remedy is well worth trying.

DR. SIGMUND AGATSTON: Did any of these patients show hypertension?

DR. W. F. DUGGAN: We have not investigated hypertension in these cases. The patients are all over 40 years of age, and I think we determined the blood pressure in only one case. All these patients responded to vasodilatation. The blood pressure has been investigated many times before, and there is a large group of cases reported in the literature.

TWO CASES OF DETACHMENT OF THE RETINA WITH TREATMENT BY MULTIPLE IGNIPUNCTURES. DR. FRANLIN BRACKEN.

CASE 1.—The retina had been detached about one month when operated on in June, 1931. The dialysis extending from the position of 6 to 8 o'clock was closed with two punctures about 13 mm. back of the limbus. The retina is completely reattached, and vision has improved from 15/200 to 20/100.

CASE 2.—The patient, operated on in October, 1931, had a much larger detachment than in case 1, with a dialysis extending from the position of 3 to 6 o'clock. Closure of the dialysis was attempted by four ignipunctures 14 mm. back of the limbus. Vision has improved from 6/200 to 20/30+, but there is a shallow detachment in the lower temporal quadrant which has remained stationary. No hole or dialysis can be seen.

DISCUSSION

DR. MARK J. SCHOENBERG: I have seen Dr. Bracken's two cases, and I think he is to be congratulated on his results. The second case, I think, is going to show an excellent result. It is doubtful if there is a hole; there may be only a thinned-out retina. Besides, the portion of the retina that looks detached is not necessarily loose and floating. There is a semisolid substance underneath the retina which anchors it to the choroid, and it is possible that the macula will remain spared.

RADIUM THERAPY IN THE NEW YORK EYE AND EAR INFIRMARY. DR. G. ALLEN, Robinson, N. Y.

Six years ago a clinic for the treatment of neoplastic lesions was established in the New York Eye and Ear Infirmary. Radium tubes and plaques amounting to 200 mg., supplemented at times with gold radon implants, have been available for the treatment of these patients. During this time 150 patients in 1,500 visits have received 900 applications of radium.

The following conditions have been treated: vernal catarrh, old trachoma, papilloma of the eyelids, angiomas of the eyelids and orbit, epidermoid carcinoma of the eyelids and eyeball and malignant tumors of the orbit.

It seems to be definitely established in the treatment of 40 patients with vernal catarrh that radium produces palliative effects well worth while. Small doses of radium are applied externally to the lids, and radium plaques directly to the lesions on the conjunctiva. Mild doses are given; so in only two or three instances has overaction occurred on the cornea. Within a week after treatment with radium improvement is noted, the secretions decrease, the pain is lessened, and the photophobia is less marked.

In epidermoid carcinoma of the eyelids radium seems to be the treatment of choice. The technic of inserting small radium needles or gold radon implants into the lesions is sound. External radiation with x-rays or radium at a distance causes overaction on the eyeball and conjunctiva, and in many instances produces permanent epilation of the cilia. The greater intensity of radiation by the interstitial method is desirable and produces the minimum effect on the surrounding tissues.

In angiomas of the eyelids radium produces satisfactory results. The endothelial cells lining the capillaries that compose angioma are radiosensitive, and the end-result is obliterative fibrosis. Papillomas and keloids are also responsive to radium therapy. The results have not been satisfactory in the treatment of gliomas and melanomas of the orbit. Metastases to the opposite eye are frequent. Primary intra-ocular tumors according to my experience do not yield to radium therapy.

DISCUSSION

DR. MARK J. SCHOENBERG: I should like to ask whether Dr. Robinson has followed the cases over a number of years, and whether he has had any cataracts as complications.

DR. G. A. ROBINSON: I have seen two cases of cataract, one in a diabetic patient, and one in a little girl who had received a very heavy dose for a sarcoma of the orbit. Mild doses not sufficient to give an intense effect on the lens or intra-ocular structures, I believe, will not produce cataract. I have followed some of these cases for ten years, and cataract is extremely rare when mild and repeated small doses of radium are used.

DR. BERNARD SAMUELS: As to radium treatment of vernal catarrh, I have not seen the slightest good result in the limited number of five or six patients so treated, whose cases I can recall.

Concerning tumors of the lids, I think what has been done by the use of radium is nothing short of marvelous. One almost never has to do plastic operations on the lids for tumors any more, as they are so satisfactorily treated by radium.

DR. ARNOLD KNAPP: I am under the impression that Dr. Robinson was pleased with his results in spring catarrh. Has he read Dr. Quick's article which appeared in the ARCHIVES OF OPHTHALMOLOGY several months ago? Does he share Dr. Quick's pessimistic views about radium treatment in spring catarrh, and has he used x-rays or radium for this condition?

DR. G. A. ROBINSON: I do not feel pessimistic in regard to radium in vernal catarrh, and I think that in 75 per cent of cases there is marked improvement of the symptoms.

I am sorry that I have not referred more cases to Dr. Samuels, or that he has not followed them more carefully, and I should take pleasure in convincing him of the value of radium in vernal catarrh. The technic is important; frequent applications are necessary. Heavy caustic treatment with beta radiations does not seem to be the best method of treatment. These cases are discouraging, and unless one has a great deal of patience it is easy to say that the results are not good.

A. DEMONSTRATION OF A METHOD OF STUDYING FACTORS INFLUENCING THE SWELLING OF THE VITREOUS.

B. CHEMICAL COMPOSITION OF THE BLOOD AND SPINAL FLUID IN PRIMARY GLAUCOMA: COMPARATIVE STUDIES.

DR. MARTIN COHEN, DR. JOHN A. KILLIAN AND DR. L. HALPERIN.

This article appeared in full in the July, 1932, issue of the ARCHIVES, page 39.

A SERIES OF RADIUM INJURY TO AN EYE. DR. ARNOLD KNAPP.

This article appeared in full in the April, 1932, issue of the ARCHIVES, page 619.

NEVUS FLAMMEUS OF THE FACE AND GLOBE: ASSOCIATED WITH GLAUCOMA, VASCULAR CHANGES IN THE IRIS AND A CALCIFIED VASCULAR GROWTH IN THE LEFT OCCIPITAL LOBE OF THE BRAIN, WITH RIGHT HOMONYMOUS HEMIANOPIA. DR. H. H. TYSON.

This article will be published in full in a later issue of the ARCHIVES.

NEW METHOD OF REGISTERING GRAPHICALLY THE OCULAR PULSE. DR. BENJAMIN FRIEDMAN.

This article will be published in full in a later issue of the ARCHIVES.

Book Reviews

Lehrbuch und Atlas der Spaltlampenmikroskopie des lebenden Auges. Part II. Linse und Zonula. By Prof. A. Vogt, Zurich. Price, 298 marks. Pp. 455, with 815 colored illustrations. Berlin, Julius Springer, 1931.

The contents of this second volume, as the preface states, are the result of collecting and selecting suitable material over a period of ten years. The conditions described were studied with the improved technic of optical section, examination with reflected light and the zones of specular reflection. Certain lenticular changes of diagnostic importance are manifested in diabetes, tetany, myotonic dystrophy, acute glaucoma, intra-ocular copper or iron particles, radiational opacity, etc.; of great importance is the information furnished by the slit-lamp on the diagnosis and prognosis of senile cataract. Similarly, in questions of accident insurance this method of examination is of great benefit. The author has also considered problems of general medical and biologic interest such as concern heredity, senile changes and injuries due to radiant energy.

The part on the lens begins with a general introduction, treating the form, the anatomic structure and certain features of the development, with comments on Gullstrand's intracapsular accommodation mechanism. In classifying the diseases of the lens, the author states that most disturbances of the lens are hereditary diseases, which include regressive senile changes, and he shows how certain definite groups of fibers are selected. Certain diseases of the lens are governed by the form of the sutures in different zones, or by the difference in the consistence of the lenticular substance, and are recognized by the yellow discoloration. Then there is a large group of cataracts, cataracta complicata, caused by exogenous factors, trauma, radiant energy, metabolic disturbances and severe ocular disease, in which the slit-lamp is helpful in differential diagnosis. The slit-lamp, furthermore, has drawn attention to the lamellar structure of the capsule of the lens and its desquamation in senile exfoliation (Vogt), which frequently leads to glaucoma, and the desquamation of the anterior lamella in glassblowers. The slit-lamp appearance of the normal lens is then described, and the peculiarities of its concentric radiating structure are explained together. The remnants of the tunica vasculosa lentis and the insertion of the hyaloid artery are fully described and illustrated.

The pathologic changes in the lens are considered under the following headings: surface deposits, congenital and early acquired changes, progressive juvenile and adolescent cataracts, presenile and senile cataract, the aging of the eye, cataracta complicata, pathology of the capsule of the lens, injuries, radiational changes and after-cataract. The final chapter takes up the normal zonula and pathologic changes in the zonula.

The enormous ground that is so admirably covered is made especially useful by the clinical features; many illustrative case histories are given, with due consideration to the literature. It is not possible to give a description of striking conditions, because every observation is instructive. It suffices to mention the exhaustive description of the many forms of embryonal axial cataract, anterior and posterior stellate cataract, cataracta centralis pulverulenta, "Spieskatarakt" and lenticonus posterior. Many of these conditions were first described by Vogt; now their description is made available, and these pathologic pictures should become the working capital of every oculist. What could be of greater importance than the recognition of the posterior saucer-shaped cataract from a practical standpoint? Now this is made easily possible with the aid of the slit-lamp. The true nature of nuclear cataract with double focus was first explained by Vogt. The author shows that certain forms of opacity are always to be found in definite lenticular zones; the cortex, adult and embryonal nuclei react at a definite time to cataract-producing conditions with the formation of different cataract. Most instructive in this connection is the schematic drawing showing the distribution of the most frequent opacities in the various zones of the lens. The important subject of cataracta complicata is admirably treated, and a new type of juvenile cataract is described in combination with neurodermatitis, a chronic disease of the skin and a cataract occurring in acute glaucoma consisting in subepithelial white spots. A thorough description of the changes of the capsule of the lens, exfoliation and lamellar desquamation, is most timely and instructive. Injuries of the lens through trauma and radiant energy, after-cataract and the subject of the zonula conclude the volume.

Nothing thus far has been said about the illustrations; they are wonderfully reproduced and are really works of art, so that the book gives not only an atlas of beautiful drawings, but a description of many important and some new conditions as revealed by the slit-lamp, which add greatly to our knowledge of ocular diseases. This work must become an indispensable part in the library of every ophthalmologist.

A. K.

Röntgendiagnostik des Schädels bei Erkrankungen des Auges und seiner Nachbarorgane. By Prof. Rudolf Thiel, Berlin. Price, 48 marks. Pp. 87, with 415 illustrations. Berlin: Julius Springer, 1932.

The x-rays have not been of much help in the study of diseases of the eyeball except for intra-ocular foreign bodies, but there are diseases of the orbit, the nasal accessory sinuses and the skull which may have the greatest influence on the eye and for which a roentgen examination is essential. These diseases of the neighborhood of the eye are described by Professor Thiel in a textbook and atlas based on personally observed cases. The subject is divided into roentgen conditions of the orbit, the nasal sinuses, the middle cranial fossa, the skull in chronically increased pressure in the brain, the teeth and the lacrimal passages. In the text, the first chapter takes up the technic of roentgen examination of the skull. In this the methods of obtaining the typical exposures are first

described, and then the following, which are necessary in diseases of the eye and its neighborhood: (1) orbit, transverse view; (2) orbit, postero-anterior view; (3) optic nerve canal; (4) nasal sinus, postero-anterior view. The corresponding part of the atlas illustrates with diagrams the position of the head to the plate and of the apparatus, and gives the resulting picture with its anatomic landmarks. Each of the various parts is then taken up in turn, and a description of the x-ray picture of the particular part in question follows. A table shows the roentgen findings and the condition of the eye in the diseases that affect that particular part. Illustrative cases are then added, giving a brief clinical history of the changes observed in the eye and in the surrounding structures, with the roentgen findings. A remarkably complete picture is thus presented in a most instructive and interesting manner. The author has collected ninety-seven cases in which he illustrates the main variations encountered in these diseases. A picture of the patient is often added and, if necessary, one of the fundus, of the field of vision and of the microscopic slide in case of a tumor. The x-ray plates are beautiful, and in their clearness and detail leave nothing to be desired. One cannot conceive of a more practical and successful handling of a clinical presentation.

Some of the interesting pictures of pathologic skulls, as in Paget's disease, myelomas and tower skulls, were obtained from the Krankenhaus in Friedrichshain with its remarkable Röntgendepartment and its unique pathologic museum. The subject of inflammation and tumors of the nasal sinuses with their secondary orbital changes is fully described. Changes in the middle cranial fossa constitute an important chapter, as the eyes are apt to be involved on account of the course of the optic nerves and the site of the chiasm. An important relation is that of the internal carotid artery and the hypophysis to the optic nerves; the optic nerves may also suffer from changes in the cranial bones and in the dura.

The author speaks of tumors of the hypophysis, parasellar tumors and calcification of the internal carotid artery (circle of Willis), giving many case histories and illustrative pictures and diagrams. In the last chapter, pseudoglaucoma in sclerosis of the internal carotid is described, with its marginal excavation, normal intra-ocular pressure and roentgen findings of sclerosis of the internal carotid artery. This change in the carotid artery was also observed in a case of disklike degeneration of the macula.

Changes of the skull in chronically increased brain pressure produce, in addition to other changes, deformities in the sella turcica. The bulging of the floor of the distended third ventricle spreads the entrance into the sella; the clinoid processes are deformed and pointed, and the attenuated floor bulges into the sphenoidal cavity. Pathologic changes in the teeth, which are important for the oculist and can be demonstrated with the x-rays, are granulomas and cysts of the roots. The illustrative cases are examples of retrobulbar neuritis and of iritis. The lacrimal passages can be demonstrated after injection of a contrast material like iodized sesame oil (from 20 to 40 per cent).

The review of this publication is not complete without again referring to the excellence of the x-ray pictures. They can have been obtained only by the most careful work of well trained technicians and with the

cooperation of the best artists dealing with the reproduction of pictures. Ophthalmology is indebted to Professor Thiel for a most useful and instructive atlas.

A. K.

Psychological Factors in Peripheral Vision. By G. C. Grindley. Report of Medical Research Council, No. IX. Price, 1 shilling. Pp. 49. London: His Majesty's Stationery Office, 1931.

Factors accounting for the great difference between peripheral and central vision are of three kinds. They are physical, such as the distortion of images from oblique rays, retinal, due to the distribution of rods and cones, and central, due to the interpretation of the images in consciousness. The present report describes the author's experiments to investigate these central factors. An apparatus was arranged in which an observer fixed a light through an aperture while objects were exposed at angles of 20, 40, 60 and 80° from the center of fixation by means of a projection apparatus. By means of a shutter, the time of exposure could be varied, but was usually one-tenth second. Six psychologists were used as observers for about 800 readings, the images exposed being drawn as accurately as the visual impression would allow. The figures used were geometric figures, combinations of circles and lines. As would be expected, the accuracy of observation decreased markedly with distance from the fixation point and with decrease in the size of the object.

Objects of the same size appeared smaller in the periphery, possibly on account of the sparsity of receptors in this portion of the retina. Some observers drew figures quite unlike the one exposed, and here apparently a previously existing "perceptional pattern" was aroused by the figure shown. Sometimes only parts of the figure were observed, indicating that certain parts dominated others in the impression formed. The same effect was noted when several figures were exposed at once, some being observed, while others were not. Where an object had been previously observed a number of times, it was seen more easily, or was wrongly seen in place of other objects not distinctly seen, indicating a "sensitization" to certain perceptional patterns.

The effect of time was tested by exposing the same object at 80° for periods of from one one-hundredth to one second. Some correct observations were made in one one-hundredth second, but the accuracy increased markedly up to one second. When other objects were exposed nearer to the fixation point at the same time, a longer time was required to recognize the same peripheral figure correctly, evidently because the attention was diverted by the more central figures. If told in which part of the field the object is to be seen, most observers see it with slightly greater accuracy, indicating the effect of a conscious effort of attention.

In experiments to determine localization in the peripheral field, most observers showed a tendency to localize objects nearer to the fixation point than they actually were.

Most of the central factors revealed by the experiments, such as those of attention, expectation and the tendency to interpret images in terms of known forms, apply to direct as well as to peripheral vision, and the author concludes that no distinct differences can be claimed in the psychic processes involved in these two types of vision.

SANFORD R. GIFFORD.

Die Röntgendiagnostik und Therapie in der Augenheilkunde. Radiologische Praktika, volume XIX. By Dr. W. Hoffmann, Königsberg. Price, 8.70 marks. Pp. 70, with 20 illustrations. Leipzig: Georg Thieme, 1932.

This is one of a series of radiologic manuals, and relates to the eye. The author is connected with a clinic that has studied particularly the effects and uses of radiant energy on the eyes, and has himself contributed to the literature on this subject. The book takes up the use of the x-rays for diagnosis and therapy in ophthalmology.

In the first part the following subjects are considered: the localization of intra-ocular foreign bodies and diseases of the eye, orbit, optic nerve and lacrimal passages. Roentgen examinations play only a limited rôle in ophthalmology compared to that in other branches of medicine. Diseases of the eye are usually readily accessible to direct observation, especially if the slit-lamp is used, while the conditions in the orbit surrounded by bone are recognized only with difficulty. An exception is the metallic foreign body, in which cases examination with the x-rays is now generally regarded as a necessity. Attempts to demonstrate foreign bodies have led to the development of a number of methods, which are all fully described. The author prefers the method of Engelbrecht, or that of Comberg. The value of roentgen examination in orbital conditions is clear, particularly in changes of the bony walls, in which the x-rays help not only in the diagnosis, but also in prognosis and in the choice of operation. As a rule, the x-rays are not of importance in conditions affecting only the soft parts of the orbit. In these the author advises the use of a Bucky diaphragm, soft rays and hard developers to give serviceable pictures with sufficient contrast. The methods and value of roentgen photography of the optic foramen are described, and the necessity of an errorless technic in hypophyseal pictures is emphasized. The lacrimal passages can de demonstrated with the x-rays after the injection of iodized poppy seed oil 40 per cent, and the topography of the bony structures about the lacrimal sac is of value in dacryocystorhinostomy.

Roentgen therapy in ophthalmology has been practiced only in recent years as possible injury to the eye was feared. Knowledge of what the eye can stand has led to satisfactory treatment of certain chronic diseases, such as tuberculosis. There is an excellent chapter on the injuries to the tissues of the eyes produced by the x-rays. These injuries may occur soon after the roentgen exposure, or several years later; they have been studied clinically and histologically by Birch-Hirschfeld. The best methods of preventing these injuries are given in a special chapter. The various conditions of the eye that have been treated with the x-rays are then taken up, with the indications, dosage and results.

The author has written a practical and comprehensive book, which is doubly welcome; the subject is important, and the book brings together information that is not easily available.

A. K.

The Experimental Study of Reading. By M. D. Vernon. Price, $3. Pp. 200. New York: The Macmillan Company, 1931.

Following a history of the experiments determining and recording ocular movements made by many observers from Javal (in 1878) to her own exact studies, described in a monograph reviewed in the December,

1931, issue of the ARCHIVES (page 966), there are chapters giving a detailed account of types and sensations of ocular movements.

Theories of visual perception, so rapid and complex and difficult to analyze, are considered, including those advanced by the German School of Gestalt psychology.

It is shown that voluntary and reflex movements about the field are in general inaccurate and erratic. To pass from one part of the field to another it was usually necessary to execute correcting movements, interspersed with fixation pauses, during which the eye reviewed its position. But in mature adult reading a series of regular and rhythmic backward and forward movements developed, the forward movements alternating regularly with fixation pauses of comparatively short and regular duration.

Cattell (in 1886) demonstrated that one does not perceive individually each letter of a word, and that one reads by word-wholes and sometimes by whole phrases at a time. Other observers support his views. The form quality of a word is determined by its length and by the number of ascending and descending letters, especially the former.

Most investigators have found that the normal frequency of fixation pauses per line indicates that one or two words are covered at each fixation. The fovea at the reading distance could contain the image from a horizontal line of only 0.6 cm. in length. At each fixation pause a very small central area is seen clearly, the surrounding area becoming more and more blurred.

Reading in children is discussed thoroughly, with an interesting description of reading disabilities, congenital word blindness, etc. Much has been written about the influence of typographic factors on the ease and efficiency of reading. There seems to be a general preference for a simple old style type, such as Caslon employed for a majority of well printed books. The length of line should not exceed 10 cm., and the printed page will always appear clearer with a moderate amount of interlineage.

In less than two hundred pages the author covers her subject thoroughly. Her style is lucid and of high literary merit. There are a full bibliography and a good index.

JOHN B. LYNCH.

Cirurgia Ocular. Volume 1. Ten Years of Specialized Surgery. By Dr. W. Belfort Mattos, recipient of Moura Brasil 1930 prize, National Academy of Medicine. Pp. 109. Sao Paulo, Brazil.

After a preface by Gabriel de Andrade, of Rio de Janeiro, Dr. Mattos described (in Portuguese) the results of ten years of specialized surgery.

A description of his up-to-date ophthalmic operating room is given, with illustrations of the usual appliances.

Then the following conditions are taken up in turn, with an outline of the preparation of the patient, the operative procedure, the indications and the results, and many illustrations: senile cataract, soft cataract, leukoma of the cornea, cyst of the iris, ocular cysticercus, glaucoma—acute (Graefe iridectomy) and chronic (Lagrange)—epibulbar tumors, pterygium, tumors of the optic nerve, trachoma, entropion and trichiasis, cicatricial obliteration of the orbit, lacrimal surgery, discission and intra-ocular foreign bodies.

A. K.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217, Faubourg St. Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66, Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Franciso Poyales, Olozaga 3, Madrid, Spain.
Place: Madrid, Spain. Time, April 16-22, 1933.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Sir J. Herbert Parsons, 54 Queen Anne St., Cavendish Square, London, W. I.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ-Wood, 12 St. John's Hill, Shrewsbury, England.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. E. W. Brewerton, 73 Harley St., W. 1, London.
Secretary: Dr. P. G. Doyne, 60 Queen Anne St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. Frederick H. Verhoeff, 82 Commonwealth Ave., Boston.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.
Place: Milwaukee. Time: June 12-16, 1933.

American Academy of Ophthalmology and Oto-Laryngology

President: Dr. S. Hanford McKee, 1528 Crescent St., Montreal, Canada.
President-Elect: Dr. B. R. Shurley, 62 W. Adams Ave., Detroit.
Executive Secretary: Dr. William P. Wherry, Medical Arts Bldg., Omaha.
Place: Montreal, Canada. Time: September 19-23, 1932.

American Ophthalmological Society

President: Dr. Edward C. Ellett, 130 Madison Ave., Memphis, Tenn.
Secretary: Dr. Emory Hill, Professional Bldg., Richmond, Va.

SECTIONAL

New England Ophthalmological Society

President: Dr. David W. Wells, Hotel Westminster, Boston.
Secretary-Treasurer: Dr. Theodore L. Terry, 243 Charles St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

President: Dr. Hans Barkan, 490 Post St., San Francisco.
Secretary-Treasurer: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Place: San Francisco. Time: 1933.

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. John H. Harter, 1215 Fourth Ave., Seattle.
Secretary-Treasurer: Dr. Francis A. Brugman, 806 Cobb Bldg., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. R. B. Smith, Alma, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

SIOUX VALLEY EYE AND EAR ACADEMY

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.
Place: Sioux City, Iowa. Time: November, 1932.

SOUTHERN MEDICAL ASSOCIATION, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Wiley R. Buffington, 211 Camp St., New Orleans.
Secretary: Dr. William D. Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Birmingham, Ala. Time: November, 1932.

SOUTHWESTERN MICHIGAN TRIOLOGICAL SOCIETY

President: Dr. W. E. McGarvey, 802 Jackson City Bank Bldg., Jackson.
Secretary: Dr. Don C. Lyons, 1405 Union and Peoples National Bank Bldg., Jackson.
Time: Third Thursday of alternate months.

STATE

COLORADO OPHTHALMOLOGICAL SOCIETY

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Ralph W. Danielson, 258 Metropolitan Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

CONNECTICUT STATE MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. M. H. Gill, 36 Pearl St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

EYE, EAR, NOSE AND THROAT CLUB OF GEORGIA

President: Dr. Thomas H. Hall, Jr., 617 Mulberry St., Macon.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. J. R. Gillum, 221 S. Sixth St., Terre Haute.
Secretary: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Royal French, 28 S. First Ave., Marshalltown.
Secretary-Treasurer: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.

MICHIGAN STATE MEDICAL SOCIETY, SECTION OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Dr. Wilfred Haughey, 303 Post Bldg., Battle Creek.
Secretary: Dr. H. O. Westervelt, 239 Pipestone St., Benton Harbor.

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Douglas F. Wood, 74 S. Ninth St., Minneapolis.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY

President: Dr. L. H. Huber, Livingston.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

NEW JERSEY STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY, OTOLOGY AND RHINOLARYNGOLOGY

Chairman: Dr. Charles H. Schlichter, 556 N. Broad St., Elizabeth.
Secretary: Dr. B. E. Failing, 31 Lincoln Park, Newark.

NEW YORK STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

Chairman: Dr. D. F. Gillette, 109 S. Warren St., Syracuse.
Secretary: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.

NORTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Axel Oftedal, 55½ Broadway, Fargo.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. H. M. Hendershott, 193 Eleventh St., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Herman A. Winkler, 224 Thayer St., Providence.
Secretary: Dr. N. A. Bolotow, 108 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. McWhorter, 106 E. Earle St., Anderson.
Secretary-Treasurer: Dr. G. M. Truluck, Orangeburg.
Time: April and December.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. W. D. Jones, 3116 Live Oak St., Dallas, Texas.
Secretary: Dr. A. F. Clark, 1034 Nix Professional Bldg., San Antonio.

UTAH OPHTHALMOLOGICAL SOCIETY

President: Dr. D. W. Henderson, 75 S. Main St., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welch, 14 Exchange Pl., Salt Lake City.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY

President: Dr. Frank P. Swart, 142 W. York St., Norfolk.
Secretary-Treasurer: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION

President: Dr. O. H. Bobbitt, Medical Arts Bldg., Charleston.
Secretary: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.

LOCAL

Academy of Medicine of Northern New Jersey, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Elbert A. Curtis, 65 Central St., Newark.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South. Time: 8:45 p. m., second Monday of each month.

Academy of Medicine of Toronto, Section of Ophthalmology

Chairman: Dr. P. J. F. Houston, 34 Hurndale Ave., Toronto.
Secretary: Dr. Alexander E. MacDonald, 151 Bloor St., W., Toronto.
Time: First Monday of winter months.

Atlanta Eye, Ear, Nose and Throat Society

President: Dr. L. C. Rouglin, 127 Peachtree St., N. E., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., Medical Arts Bldg., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

Baltimore Medical Society, Section on Ophthalmology

Chairman: Dr. Harry Friedenwald, 1212 Eutaw Pl., Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. D. Andrews, 33 Allen St., Buffalo.
Secretary-Treasurer: Dr. Thurber Le-Win, 112 Linwood Ave., Buffalo.

Chicago Ophthalmological Society

President: Dr. Frank Brawley, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medical and Dental Arts Club. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. M. W. Jacoby, 2323 Prospect Ave., Cleveland.
Secretary: Dr. H. C. Rosenberger, 1065 Rose Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. Roy B. Metz, Hanna Bldg., Cleveland.
Secretary: Dr. Paul G. Moore, Medical Arts Bldg., Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. L. F. Appleman, 308 S. Sixteenth St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. John B. Alcorn, 167 E. State St., Columbus, Ohio.
Secretary-Treasurer: Dr. Edward W. Harris, 681 E. Broad St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. O. M. Marchman, 1717 Pacific Ave., Dallas, Texas.
Recording Secretary: Dr. W. Mood Knowles, 1717 Pacific Ave., Dallas, Texas.
Corresponding Secretary: Dr. J. M. Potts, 1717 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. Cecil C. Jones, 1121 Equitable Bldg., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. R. H. Seeley, 9 Court St., Rutland, Vt.
Secretary-Treasurer: Dr. Arthur F. Holding, 142 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. R. H. Needham, 1304 Medical Arts Bldg., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1011 Medical Arts Bldg., Fort Worth.
Place: University Club. Time: 6:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 26 Sheldon Ave., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. L. W. Raney, 609 Fannin St., Houston, Texas.
Secretary: Dr. J. F. Gamble, 201 Main St., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., first Thursday of each month from September to June.

Indianapolis Ophthalmological and Oto-Laryngological Society

President: Dr. John W. Carmack, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. Albert N. Lemoine, 906 Grand Ave. ,Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

President: Dr. Ferris L. Arnold, 923 Security Bldg., Long Beach, Calif.
Secretary-Treasurer: Dr. Clyde E. Harner, 312 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from September to June.

Los Angeles County Medical Society, Section on Ophthalmology and Oto-Laryngology

President: Dr. J. Frank Friesen, 1208 Roosevelt Bldg., Los Angeles.
Secretary-Treasurer: Dr. F. H. Brandt, 1052 W. Sixth St., Los Angeles.
Place: Mary Louise Cafe. Time: 6:30 p. m., fourth Monday of each month from October to May, inclusive.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. Albert P. Tibbets, 1801 I St., Washington.
Secretary Dr. G. Victor Simpson, Rhode Island Ave., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Oto-Laryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month. Annual meeting, Jan. 10, 1933.

Milwaukee Oto-Ophthalmic Society

President: Dr. T. F. McCormick, 324 Wisconsin Ave., Milwaukee.
Secretary-Treasurer: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., third Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. P. E. Bousquet, 66 Sherbrooke St., West, Montreal, Canada.
Secretary: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Hilliard Wood, 700 Church St., Nashville, Tenn.
Secretary-Treasurer: Dr. H. C. Smith, Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. Charles A. Bahn, 150 Baronne St., New Orleans.
Secretary-Treasurer: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Mark J. Schoenberg, 1160 Park Ave., New York.
Secretary: Dr. Algernon B. Reese, 73 E. Seventy-First St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. L. G. Howard, 532 First Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. W. A. Cassidy, 1620 Medical Arts Bldg., Omaha.
Place: University Club, 1914 Harney St., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Secretary: Dr. R. W. Vaughan, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

Chairman: Dr. E. S. Amsler, 53 S. Fitzhugh St., Rochester, N. Y.
Secretary-Treasurer: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. Julius H. Gross, Carleton Bldg., St. Louis.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Charles J. Boehs, Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. D. Dumas, 425 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: 8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert C. Martin, 384 Post St., San Francisco.
Secretary: Dr. George N. Hosford, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Third Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. J. L. Scales, Medical Arts Bldg., Shreveport, La.
Secretary-Treasurer: Dr. W. L. Atkins, 505 Medical Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 7:30 p. m., first Monday of each month from October to June.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. David Hartin, Old National Bank Bldg., Spokane, Wash.
Secretary: Dr. W. W. Henderson, Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. W. G. Dickinson, 504 State Tower Bldg., Syracuse, N. Y.
Secretary-Treasurer: Dr. Harold H. Joy, 504 State Tower Bldg., Syracuse, N. Y. N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, 108 W. Sixth St., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, 108 W. Sixth St., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.